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EDITED BY

LIONEL S. BEALE.

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that it was not possible to make out the ultimate distribution of the nerve fibres in recent specimens examined in water, serum, vitreous humour, and the like (1859). After performing many experiments I was compelled to accept the conclusion, that such minute anatomical points could only be demonstrated in specimens immersed in syrup, glycerine, or some such viscid, highly refractive material, miscible in all proportions with water. By this means I was enabled to subject the section to very great pressure, and reduce it to the tenuity required for examination by the highest powers (more recently $\frac{1}{25}$ and $\frac{1}{50}$) without destroying the most delicate structure. By comparing uninjected and injected specimens of the very same tissues, I have proved that capillary vessels may be readily mistaken for fine nerve fibres, and that when shrunk and very much stretched, it is not possible to distinguish them as capillaries at all. In this state they often appear like fibres of connective tissue.

I have also proved that when the transparent injecting fluid, which contains glycerine and a little chromic acid, is introduced into the vessels immediately after death, it permeates the thin capillary walls, and preserves and hardens slightly, all the delicate tissues, including the nerves. In this way alone can a highly refracting fluid be uniformly distributed to all parts of a tissue immediately after the death of an animal. To this mode of preparation I was gradually led many years ago, after trying very numerous experiments.

Next I would respectfully submit that those who correct me will insist that the nerves stop short at a point a very short distance from the dark-bordered fibre, although I have stated over and over again that in my specimens they may be followed a long way beyond this point. In fact my correctors will make the nerves end a very short distance from the point where I conceive the really important part of a most extensive plexus system of fine nerve fibres only begins. The finest nerve fibres distributed to the muscles of the frog for example, are far finer than is supposed in Germany, and the fibres delineated by Kölliker, Kühne, Engelmann and others as single fibres, really consist of several fine fibres. Kölliker admits the existence of pale nucleated intermuscular fibres, first described by me, and agrees with me in the opinion that they lie upon the external surface of the sarcolemma. According to him the delicate sheath is prolonged upon the pale fibres, and he infers that here and there at least a thin layer of the white substance extends along the pale fibre. Kölliker very rightly distinguishes the nuclei of the pale fibres (*c*) from the nuclei of the muscle (*d*) (Plate XI, fig. 2), while Kühne had mistaken the latter

for his peculiar 'nerven-end-knospen.' But, in support of his former views, Kölliker insists that the pale nerve-fibres terminate by free extremities outside the sarcolemma.

Kölliker regards the ends he represents as real natural terminal extremities, but I think that his own drawings contradict his conclusions, for the abrupt ends represented by him are really much more like accidental breaks than true terminations. In my own observations upon the breast-muscle of the frog, the nerve-fibres are also represented as ceasing (fig. 3, Plate XI); but I feel quite sure, from observations upon other specimens, that they continued farther. I could not follow them in particular instances, in consequence of the extreme delicacy of the nerve-fibres, and the thickness and refractive power of the large muscular fibres upon which they ramified. My own specimens are figured in Plates XI, XIII, and XIV, also from the breast-muscle of the frog.

I shall endeavour to show (see page 175) that the lines which Kölliker supposes to be the outline of the sheath, result, in many instances, from the presence of very fine nerve-fibres, which form, with the fine fibres resulting from the division of the dark-bordered nerve-fibre, fine compound nerve-fibres which are distributed upon the surface of the sarcolemma as a network or plexus. Compare the nerve-fibres in Kölliker's figure copied in Plate XI, fig. 2, with my figures 2, 3, 4, Plate XIII, and figs. 7 and 9, Plate XIV.

The main difference then between the distribution of the nerves upon the elementary muscular fibres of the breast-muscle of the frog, and those of the diaphragm of the mouse, described in my paper published in the Phil. Trans. for 1860, consists, according to my observations, in the size and number of the nerve-fibres distributed to a given amount of contractile tissue. In the frog the difficulty of demonstration results from the extreme fineness of the nerve-fibres, which ramify upon the sarcolemma, and from the very few nuclei, as compared with those in connection with the nerves of the mouse. The distribution in essential points is the same, and my latest observations confirm the results obtained in my first paper in all essential particulars. The nuclei of these nerve-fibres, and the fine fibres themselves, have been considered by many German observers as belonging to the connective tissue beneath which they lie, but no one in Germany has as yet attempted to discuss this part of the question in detail, indeed, from the observations made by some, it would seem that they had neither patience nor inclination to enter upon it.

The nerve-fibre, represented by Kühne as passing through

the sarcolemma of the muscular fibre of Hydrophilus, consists of a bundle of very many fibres which lie upon the surface of the sarcolemma, and of branches of tracheæ which are not delineated in his drawings. There is no single nerve-fibre in the insect nearly so wide as delineated by Kühne, and what he terms the axis cylinder of one nerve consists in all probability of several entire nerve-fibres. The bundles of fibres delineated by him in his Plate II., figs. 9 and 10, as ending in the elementary fibre are, I think, a considerable distance from their finest terminal branches.

It is not possible to see many of the finer bundles of insect nerves in the mode of examination adopted by Kühne. In the last number of the Microscopical Journal (October, 1864) will be found a paper by me upon the structure of the sarcolemma, and the arrangement of the nerves in insect muscle, in which this question is considered.

The peculiar bodies described as 'nerven-end-knospen' are in some cases the nuclei of nerves upon the sarcolemma, in others the nuclei of the contractile tissue beneath the sarcolemma. With regard to the very peculiar structure of those represented in the drawing, in Kühne's plate, in his memoir 1862,* I can offer no opinion, as I have never seen anything at all approaching the representation.

I believe that Kühne's pale terminal fibres really consist of bundles of very fine fibres which divide and subdivide into still finer compound fibres, which are so very fine as not to be visible in specimens as usually prepared.

I might criticise many of the drawings separately, but will only offer the opinion that the dark-bordered fibres made to terminate in fig. 12, A. B. C., Plate II., are a long distance from their finest subdivisions.

Kühne has recently reiterated his conclusions in another paper (Ueber die Endigung der nerven in den Muskeln. Virchow's Archiv. Band 27), which is illustrated with several drawings. I regret that I have not been able to confirm the conclusions at which he has arrived in the memoir. Nor have I ever been able to see the nerves in fresh muscles with the distinctness given in his drawings. I have seen the numerous cells or nuclei represented near the dark-bordered fibre, as shown in his figures, but they were arranged more regularly in my specimens, and in lines, and between them were very pale granular fibres. They were on the surface of the sarcolemma, and as the vessels were injected I could demon-

* By a misprint in the note on p. 162 the date of this memoir is stated to be 1860 instead of 1862.

strate very positively which nuclei belonged to the nerves and which to the capillaries. I have stripped off from the fibre such collections of cells, leaving the sarcolemma with its contents behind. I can assure Kühne that I have traced fine pale fibres from dark-bordered fibres, not so wide as those represented in fig. 11 of his plate, over thirty or more elementary muscular fibres, in the mylo-hyoid muscle of the Hyla (see Plate XII), and that what he terms the nuclei of the nerve-sheath are, in many instances, the nuclei of fine nerve-fibres running in the sheath, which are much nearer to their terminal distribution than the dark-bordered fibres which he makes to end beneath the sarcolemma. See fig. 1 at *d*, figs. 2, 4, 6, Plate XIII.

But it is necessary for me to enter more into detail with regard to the arrangement described by Kühne, in his last two papers, for it will be noticed that in these last communications, appearances very different to those advanced in the original memoir are delineated.

From the paper in the 27th volume of Virchow's Archiv, Plate XI., I have selected certain figures, 1, 2, 3, and 4, from the rabbit, and fig. 7 from the dog. The most important parts of these figures have been copied in Plate XI., figs. 4, 5, 6, 7.

With regard to the fig. 1 copied in Plate XI., fig. 6, I am of opinion—1. That the nerve never ends abruptly in any case, as represented; 2. That the nuclei represented as beneath the sarcolemma are really upon it, that is external to it; 3. That these nuclei have been disarranged in the process of preparation.

With regard to his fig. 2 (copied in Plate XI., fig. 7), I would remark in addition, that the dark line which represents the outline of the white substance is continued as the outline of the sarcolemma, an arrangement which most certainly does not exist in nature.

In Kühne's figs. 4 and 7 (copied in Plate XI., figs. 4 and 5), the dislocation of the nuclei is clearly represented.

The 'nervenhügel' H, delineated in these drawings, are not represented in Kühne's earlier papers. In the four drawings, *a* is considered by Kühne as the nuclei of the nerve sheath, but (at least in some instances) these are nuclei of fine nerve-fibres running in the sheath, as already stated.

Kühne has not represented the capillaries in any of his drawings. The explanation of the appearances represented by him in the nervenhügel, is seen by reference to my fig. 8, Plate XXIII., Phil. Trans. 1860, in the upper part of which is just such a collection of nuclei with the nerve-fibres, in which they

are imbedded. In Kühne's drawings the fine nerve-fibres are disintegrated, and the nuclei completely disarranged.

The appearances represented in these 'nervenhügel' of Kühne were familiar to me in the mouse, before I arrived at the conclusions published in my first paper, and at this time I succeeded in demonstrating that the arrangement of the 'hügel' was not natural.

In Plate XI., fig. 9, the points to be seen upon the surface of the sarcolemma of a muscular fibre from the diaphragm of the white mouse are represented, and in fig. 10 a portion is shown much more highly magnified. The nuclei are connected with nerve-fibres, which lie outside the sarcolemma. In fact vessels and nerves can be stripped entirely away from the sarcolemma of the muscular fibre of the white mouse, leaving this membrane entire. Had this specimen been prepared according to the methods in ordinary use, all that would have been observed would have been a cluster of nuclei, irregularly arranged, like those in the nervenhügel.

Further, the mode of formation of the nervenhügel, so distinct in the insect muscle, will be seen by reference to my paper, just published in the Microscopical Journal.*

But it must not be supposed from these remarks, that I altogether deny the existence of the expansion of the nerve over the elementary muscular fibre, as delineated in this and in Kühne's last paper. Far from this, I readily acknowledge that such an appearance does exist, and I have beautiful specimens showing distinctly some of the appearances in question, but the structure is undoubtedly outside the sarcolemma, and my interpretation is very different from that given by other observers who have seen the appearance in question,—but this shall be fully considered in another memoir.

In the paper, "Die Muskelspindeln" (Virchow's Archiv., vol. xxviii.) Dr. Kühne has ventured to represent a dark-bordered fibre, close to its supposed intramuscular termination, three-fourths as wide as the muscular fibre itself! Part of this figure I have copied in Plate XI., fig. 8. In a former paper he has represented as one nerve a bundle of fibres, which really crosses over or under the muscular fibre, and probably divides into smaller bundles of finer fibres, which pursue a course of some length before they reach their ultimate distribution.

Kühne, however, supports his former opinion in yet another

* On the structure and formation of the sarcolemma of striped muscle, and of the exact relation of the nerves, vessels, and air-tubes (in the case of insects), to the contractile tissue of muscle. Trans. of the Mic. Society. See also Figures 4 and 5, Plate XV., of this journal.

paper (Der Zusammenhang von Nerv und Muskelfaser, Virchow's Archiv., vol. xxix). The illustrations accompanying this paper represent the nervenhügel and their connection with the muscular tissue, as seen in a transverse section of the elementary muscular fibres of the white rat and mouse. I have had these figures copied in Plate XV., figs. 1 and 2. They explain themselves. I may remark, with reference to these figures, that in my own specimens from the white mouse, the dark-bordered fibres certainly do not terminate as Kühne has represented, and that exceedingly pale granular fibres can be followed for a considerable distance beyond the point where the highly refractive white substance ceases (see figs 9 and 10, Plate XI., in which the capillaries are also represented).

In Plate XV., fig. 3, I have copied (but reduced to half the size) Kühne's last representation of the arrangement of the nerve plates and the formation of the nervenhügel. The axis cylinder is here made to expand into a broad 'end plate.' The oval nuclei near the expansion of the axis cylinder are the nuclei of the 'nervenhügel.' The nuclei of the sarcolemma are marked. I must beg the reader to compare the nerve 'end-plate' in this figure with the terminal pale fibres, &c., 'nerven-end-knospen' figured in Plate XI, fig. 1, published in the last number of the Archives. (Taf. ix. Virchow's Archiv., Vol. XXX.)

The reader will have noticed, that there is a broad and essential difference between continental observers and myself in reference to the distance which fibres forming the structural continuation of the dark-bordered fibre may be followed beyond the point where the dark-bordered fibre ceases. Kühne, Cohnheim, Rouget, Krause, Engelmann, make the nerve end a very short distance from the point where the dark-bordered fibre ceases, and the latest researches make the dark-bordered fibre end abruptly in its terminal end-plate, so that there is not even a pale fibre as long as that of the Pacinian corpuscle. Kölliker, on the other hand, traces pale fibres for some distance in continuation with the dark-bordered fibre and on the external surface of the sarcolemma. So far, in essential points, Kölliker coincides with me, but I stated that the fibres did not end as Kölliker supposed, but might be followed for long distances as excessively fine fibres only demonstrable, however, in specimens prepared in a particular manner.

It was to be supposed that these pale fibres which I have described as nervous, would be characterised as fibres of connective tissue, by opponents who have not seen my specimens, and especially by those who have not examined my drawings, or

carefully perused my observations. This is at the same time the obvious and the most easy mode of attack, since it is not possible for me to show these fibres to every one, and any assertion I may make with regard to them, such, for example, as, 'that I have traced them onwards from the dark-bordered fibre,' may be doubted and set down as an error of observation or as resulting from the mode of examination employed, &c. For years it was maintained that 'Remak's fibres' were connective tissue, and the doctrine is still held by some German observers. There are persons who look upon branches of the sympathetic as modified connective tissue, in which 'apolar' nerve cells are embedded, and who do not consider what such fibres are for, or what purpose is fulfilled by such cells. There are observers who seem to think that connective tissue is the most important texture in the body, that it has special canals for its nutrition, special corpuscles for its formation, that it supports higher tissues in position, that it is the principal, if not the sole, seat of some of the most important pathological changes.

It has often happened that the mere dictum of one observer has been permitted for years to outweigh the facts ascertained by the actual observation of others, and it is natural, and, indeed, right, even in matters of mere observation, that a high authority having a well-earned position as an observer and a high reputation as an author, should influence the opinions of others, and, indeed, exert very considerable weight.

At the same time it will be conceded by the highest authorities themselves, that good observers have been led to entertain erroneous views, and there is no department of natural knowledge in which such conflicting statements have been made upon mere matters of fact and observation as in minute anatomy. I must therefore press my own observations upon the attention of observers, not as having authority or reputation, but simply as an observer who has at least spared no time, pains, or trouble during many years to ascertain the truth regarding the mode of distribution of nerves to muscle.

In several papers and in many drawings I have endeavoured to show that the fine fibres I have described as nerves are structurally continuous with dark-bordered fibres. I have stated that the specimens may be examined by any observers who desire to see them. I have shown that fine fibres agreeing in every respect with those I have described in muscle, exist in other tissues to which nerves are distributed, and many of these are admitted by all who have seen them to be nervous. In Plate XIII., fig. 1, at *d*, is a fibre which would be called con-

nective tissue by some, continuous with a dark-bordered fibre. In fig. 2 are 'connective tissue fibres' (?) running on one side only of a dark-bordered fibre, giving off branches which further divide and sub-divide and form a plexus or network of very fine "connective tissue fibres!" In the other figures in this plate fibres of the same kind are seen, and their continuity with undoubted nerve fibres, either at the spot copied in the drawings or at a short distance from this, was demonstrated. Corresponding fine fibres are represented in Plate XIV.

Then there is collateral evidence in favour of the conclusions, which would, I think, be drawn by any unprejudiced person from a careful examination of several of my specimens, and yet, in spite of all, Kühne reiterates, the old cry, 'connective tissue,' 'vascular nerves!' No arguments are advanced, no specimens to explain the facts in another way made, nay, specimens are not even prepared, according to the method I have stated to be necessary, to see the fine nerve-fibres with the very extensive distribution I have described. While it is certain that plexuses and networks exist, is it not possible that 'Endplatten,' 'Nerven-end-knospen,' 'Nervenhügel,' &c., may exist as well (?), but Kühne is not contented with establishing the existence of the structure he has described, but proceeds to state that networks and plexuses described by me are not to be demonstrated, although I have seen them, and have shown them to other observers.

I would ask:—will observers accept the conclusion, that long elementary muscular fibres are influenced by the nerves at only one single point in their length, when dark-bordered and fine fibres cross them at short intervals throughout the entire length of the muscular fibre, as any one can demonstrate in the muscles of the eye of the frog, or, better, the cameleon, or in the diaphragm of the mouse, or in the very thin mylohyoid of the frog? For what purpose do the nerves cross the muscular fibre at many different points and pursue a most circuitous course, if a nerve-fibre is to become connected with it at one spot only, and influence the muscular fibre at this single point?

Those who hold that the plexuses of coarse and fine fibres which are to be demonstrated in the case of every muscle are an arrangement not connected in any way with the action of the nerve upon the muscle, should show not only why nerves invariably exhibit this arrangement, but why the nerve-fibres do not at once pass to the point of the muscular fibre with which they are connected, by the shortest and most direct route.

Will observers accept the conclusion that the relation of

nerves to striped and unstriped muscular fibre is essentially different, and that the unstriped fibre receives a more abundant supply of nerves than striped muscle? Will anatomists accept the fact that nerves in tissues generally form networks and plexuses of exceedingly fine fibres, without doubt,—and the statement that in this one tissue—voluntary muscle, they terminate in end plates in contact with the contractile tissue? Is it probable that the muscular fibres of the heart should be supplied with nerves upon a principle totally different to that which obtains in other striped muscles, or that some muscles of the tongue should be supplied by nerve networks, and others by nerve plates as must be the case, unless it be shown that the finest muscular fibres in the tongue, the diameter of which is many times less than that of the smallest nerve-plate figured, nay, less than the terminal portion of many of Kühne's dark-bordered fibres—are not supplied with nerves at all, although they contract like other striped muscles?

Will it be accepted as a fact, that the terminal nerve-fibres in the muscles of the trunk and limbs are many times the diameter of the terminal fibres in other tissues, and that a very long muscular fibre is influenced in its entire length by an end organ at one or other extremity, or in its centre? Can it be supposed that it is necessary for the nerve tissue to come into absolute contact with the contractile tissue of the muscle while nerves are admitted to influence other tissues without being in structural continuity with them?

Will observers accept the conclusion that the nerve perforates the sarcolemma of voluntary muscle before any observations have been offered upon the manner in which it is developed in such a locality? If the views which are now advocated by Kühne and others, with regard to the arrangement of nerves in muscle be true, it must follow that muscle forms a special exception to every other tissue in the body, both as regards the mode of distribution of its nerves, and the manner in which nerves influence the tissue. Will anatomists accept these views as affording the true account of the distribution of nerve to voluntary muscle, in the face of the fact that neither the vessels nor the nuclei connected with them are to be seen in the specimens advanced as demonstrative of the doctrines most in favour in Germany? Is it probable that a plan of demonstration which fails to render the vessels and other structures distinct and clear, should render definite a structure of such far greater delicacy than vessels, as the pale nerve-fibres? On the other hand, is it not reasonable to conclude that a plan of demonstration, which it is admitted displays most clearly the

finest nerve-fibres in tissues generally, will enable us to demonstrate them in muscle?

The arrangement of the terminal branches of the nerve in voluntary muscle advanced by Kühne and others, if true, is peculiar to this tissue alone. There is no other tissue in which nerve-fibres have been shown to terminate in end plates of such remarkable structure, and the most positive demonstrative evidence is required before such a deposition can be accepted as the normal and essential arrangement necessary for the action of the nerve upon the muscle, in the face of collateral evidence of the strongest character.* This collateral evidence, and the positive observation in innumerable specimens of a very different arrangement, are met by the mere assertion that the nerve-fibres seen are not nerve-fibres, and are composed of gelatin, or if nerves, they are vascular nerves.

I would answer simply that the fibres in question are of the same physical and chemical character as the fibres admitted to be n e r v o u s in the cornea, the bladder and mesentery of the frog, and elsewhere. They are structurally and directly continuous with dark-bordered fibres. Moreover, in the same specimen, the vessels, the vascular nerves, and the motor nerves are all demonstrated, and not only so, but the course of the fibres can be followed over a very wide area, and, oftentimes, fibres as fine as those in the cornea can be followed from one undoubted nerve-fibre, across many elementary muscular fibres to another trunk.

With reference to the points urged by Kühne and those who support him, I would remark that no answer has been given to the obvious objections that may be made to the interpretation he has given of the appearances he has seen. For instance, it may fairly be said—

1. That the nerves he gives as penetrating the sarcolemma really lie over or under the muscular fibre he delineates.†

2. That the irregular arrangement of the nuclei represented by him results from derangement, and does not exist in nature.

* I wish it to be clearly understood that I do not deny that the appearances described have been seen and are to be demonstrated, but I think that they have been probably interpreted wrongly, and I am led to the conclusion, which is supported by very strong evidence, that the view in question does not represent the mechanism through which nerve as a general rule influences striped muscle.

† I have remarked that one very important advantage gained by the use of very high powers ($\frac{1}{25}$ and $\frac{1}{50}$ magnifying from 1,500 to 3,000 diameters linear) is that structures apparently upon the same plane are demonstrated to be on very different planes. In muscle a nerve-fibre often appears by a twelfth to be situated beneath the sarcolemma, while the $\frac{1}{25}$ or $\frac{1}{50}$ show it to be situated beneath the muscular fibre itself.

While it is to be remarked—

3. That he has never seen the fine branches of the nerve-fibres which are to be demonstrated by other processes of investigation.

4. That many of the nuclei considered by him and others as nuclei of the nerve-sheath are really the nuclei of fine nerve-fibres which run in the sheath. These fine nerve-fibres are figured in several of my figures. I would especially draw attention to figs. 1 and 2, Plate XIII.

I will here record the brief criticism passed upon my observations by a French anatomist, M. Rouget, "Il est facile de s'assurer que la description de Beale est completement inexacte et ne saurait arrêter longtemps un observateur exercé et attentif." (Brown-Sèquard's Journal, 1862)!

It would be superfluous to reply to these remarks, which not only settle the question at issue, but render nugatory any conclusions to which I may be led by future work. All that remains for me is to commend the observations of M. Rouget to the attention of every 'observateur exercé et attentif.'

It is unnecessary for me to reply specially to the views of Krause, Margo, Colnheim, Aeby, Engelmann and others, for the remarks I have offered really form an answer to their conclusions, which, like Kühne's, are completely at variance with mine. Moreover, Kühne has recently given an outline of their conclusions in the 30th volume of Virchow's Archiv. The sketches illustrating his remarks are rather rough, and the one he has inserted as an illustration of my view is likely to cause rather an erroneous impression as to the course actually taken by the nerve-fibres as they traverse the muscles. This diagram is rendered still more confused by the attempt to give Kölliker's view as well as my own in one drawing. In a controversy of this kind observers should endeavour to give accurate representations of the views of their opponents. If my friend Kühne refers to my conclusions at all, it is but just he should give his readers a clear account of the views which I hold.

I now propose to offer some further remarks upon matters of importance already referred to in my memoirs, but which have not been noticed by those who have criticised my observations. Some of these observations seem to me of considerable importance, because they afford valuable collateral evidence in favour of my general conclusions.

Of the fine Nerve-fibres of the Sheath.

The nuclei represented by Kühne and Kölliker as the nuclei

of the sheath of the nerve, are clearly seen in my specimens, mounted in highly refracting fluids, to be nuclei of fine nerve-fibres running in the sheath. Kölliker, in some of his drawings, has represented the dark-bordered fibre as ending within the sheath, while the latter is prolonged onwards for some distance, and then made to terminate abruptly, or in a more or less rounded extremity.

The different and incompatible views existing between continental observers and myself are in some measure due to this sheath question. The so-called sheath is not a 'tube' or 'membrane,' or 'tubular membrane,' which contains the other constituents of the nerve-fibre; nor is it a sheath which invests them, but it is simply a transparent matrix, in which nerve-fibres, coarse and fine, are embedded. The so-called sheath is not formed as a special structure to invest the nerve-fibres, but it results from changes occurring in the nerve-fibres themselves. This 'sheath,' or 'tubular membrane' of the so-called dark-bordered fibre precisely corresponds to the transparent connective tissue, in which the fine nerve fibres are imbedded. It is a form of connective tissue, and in many situations where nerves existed at an earlier period, nothing but this so-called sheath remains. All the soluble fatty matters have disappeared, and this material, which is not readily absorbed, is left behind. Vessels may waste, and ducts of glands may waste, and leave behind the same sort of transparent connective tissue. Moreover, as I have before stated, it is altogether a fallacy to suppose that near the peripheral distribution, every single branch of nerve-fibre is surrounded by its own separate sheath. The fact is, that several branches are imbedded in the same band or cord of transparent connective tissue, and fibres from one side of such a band often cross over to the opposite side, and in very many instances one fibre pursues a spiral course around another.* There may be one or two dark-bordered fibres, and five or six very fine fibres, embedded in the same matrix (Plate XIII., fig. 3). The fine fibres, in many cases, may be most positively proved to be continuous, with dark-bordered fibres. It is almost certain that the great majority of these 'fine fibres of the sheath' are continuous with dark-bordered fibres. A very distinct instance of this is represented in Plate XIII., fig. 1.†

* See "New observations upon the structure and formation of certain nervous centres," 1864. Plate VI., Fig. 29.

† The engraver has, unfortunately, cut away from fig. 3, Plate XI., several of the fine lines, regarded by me as fine nerve fibres. The power under which this specimen was examined (700 diameters) is too low to see them very distinctly in many specimens, and it often happens that these delicate fibres are destroyed in preparing the specimen.

Doubtless some of my opponents will still cleave to the idea that these fine fibres in the sheath are not nerve-fibres at all, but the evidence is most conclusive, and I am quite ready to discuss the question at a greater length than I have done hitherto. I will only say here, that if the fine fibres which I have figured are not nerves, it follows absolutely that there are no fine terminal nerve-fibres at all, and all nerve-fibres terminate in connective tissue. The view that no fibre is a nerve-fibre unless it be dark-bordered, cannot any longer be entertained, for in no peripheral tissue or organ that I have studied does the really active part of the nerve-fibre commence, until the dark-bordered character has ceased. Where the dark border ends is not where the nerve ends, but it is where the extensive active peripheral nerve plexus commences. If all the fine plexuses demonstrated in muscle are not nervous, the plexuses of precisely the same character in the bladder of the frog, in the cornea, and other tissues, admitted by my opponents to be of this nature, are not nervous. I conclude no one would maintain that neither the fine fibres I have described in muscle, nor those in the bladder, &c., are nerve-fibres.

It has, then, been shown that at least in very many cases the fine granular fibre which invariably accompanies the fine terminal portion of the dark-bordered fibre is continuous with a dark-bordered fibre in the trunk.

This is the case in motor nerves distributed to muscle (pectoral, mylohyoid; hyla), and also in the nerves distributed to sensitive surfaces (palate, frog).

And it has been shown that a fine dark-bordered fibre often divides at a certain point into *a*, a dark-bordered fibre, and *b*, a bundle of fine fibres.

It has been shown that dark-bordered fibres seldom run alone. I have so seldom seen a fine single dark-bordered fibre which has not been accompanied by a pale, granular fibre, which invariably pursues a spiral course round it, that I am inclined to think that all dark-bordered fibres near their destination are accompanied by fine fibres.

And it has been shown that at the peripheral distribution fine granular fibres (probably composed of several finer ones) do run alone, that is without dark-bordered fibres, forming networks. Many of these very fine, pale, granular fibres are not more than the $\frac{1}{10000}$ of an inch in diameter, but there is reason to conclude that even these are composed of still finer fibres.

And it has been shown that the fine granular fibre running by the side of the dark-bordered fibre in certain cases unites

with the bundle of fine fibres, forming the continuation of the latter, to form a common, fine granular trunk, composed of very many fine fibres.

From many facts demonstrated, it seems probable that nerve-fibres, as they pass to their distribution, divide into a vast number of separate branches, which, after pursuing a course over the tissue to which they are distributed, of some extent, pass into other nerve trunks, and thus return to the cells of the nerve centre from which they started.

Nerves Distributed to the Elementary Fibre throughout its Length.

It should be particularly borne in mind that the differences between Kölliker, Kühne, Margo, Engelmann, Rouget and many others, and myself, with reference to the supply of nerves to voluntary muscle are very wide. Although the above observers differ from each other as to the precise relation the nerve bears to the contractile tissue of the elementary fibre; they all agree that the nervous supply to each individual elementary fibre is limited to a very small portion of its entire extent. Some indeed think it doubtful even if every fibre of a muscle is supplied at all, believing that only a small part of the entire muscle is brought into any very near relation with the nerve-fibres. Now, although I have not demonstrated the fact so distinctly as it is doubtless capable of being demonstrated, upon the large elementary muscular fibres of the frog, I am convinced that even in them there exists a network of very fine nerve-fibres, with nuclei here and there all over the surface of the sarcolemma, from one end of every elementary muscular fibre to the other. Doubtless, in some portions the meshes are finer than in others, and the fibres are much more numerous, but everywhere very fine but faint, and exceedingly delicate fibres are occasionally to be demonstrated. They much resemble the fine fibres in the bladder, in the cornea, and the mesentery of the same animal.

Nor have the above observers described the very numerous dark-bordered nerve-fibres which cross the muscular fibres at comparatively short intervals, as, for example, in the muscular substance of the tongue, the little muscles of the eye, over which there may be seen a complete network of dark-bordered fibres (remarkable in the muscles of the eye of the cameleon).

I have repeated my observations upon the muscles of the white mouse, and feel certain that the representations I have given are correct, although the execution of the drawings is

somewhat too rough and coarse to give a correct idea of the actual appearances observed.

It should be remembered that in these specimens the vessels are injected, and the capillaries form a very fine network over the surface of the muscular fibres. As I have stated, in the intervals, very fine granular-flattened nerve-fibres may be seen. Nuclei can be seen in the capillary walls, and nuclei in the fine nerve-fibres, and, besides these, the nuclei of the muscle. In fact the elementary fibres of the diaphragm of the white mouse are almost entirely covered with nerves and capillary vessels.

Opponents have asserted that the nuclei I have described are the nuclei of the connective tissue. In reply, I affirm that not only is much of their 'connective tissue' composed of nerve fibres but they have mistaken the nuclei of capillaries, as well as those of nerves, for connective tissue corpuscles.

Of the Sentient Nerve-Fibres of Voluntary Muscle.

It has been inferred by Reichert, and the opinion has been supported by Kölliker, that certain nerve-fibres distributed for the most part upon the surface of the flat thin breast-muscle of the frog are sentient in their nature. These so-called sentient fibres are stated by the above observers to be derived from one principal 'sentient fibre,' in the same trunk with the purely motor nerve-fibres, and Kölliker states that these sentient fibres branch out over the whole extent of the muscle, and may be seen even in parts which are destitute of 'motorial nerves.'

What I have been able to observe, with regard to the distribution of the bundle of dark-bordered fibres upon the breast-muscle of the frog, does not convince me that the above view regarding the sensitive function of these fibres is true. What I have seen is as follows :—

The bundle of nerve-fibres passing to the breast-muscle, as it crosses the muscle divides into several smaller bundles resulting from the subdivison of the primary nerve-fibres which pass to different parts of the muscle, from these minor bundles many nerve-trunks pass, which at length divide and subdivide upon the surface of the sarcolemma of the nearest elementary muscular fibres, while some pursue a much longer course, passing between muscular fibres, or ramifying in the connective tissue upon the surface of the muscle. There are in fact—

1. Dark-bordered fibres which divide upon the surface of the sarcolemma, and in contact with it, and,

2. Dark-bordered fibres which may be traced over and amongst very many muscular fibres for a long distance.

The fibres of the first set appear to lose themselves upon the sarcolemma, the last often seem to be lost amongst the connective tissue. The first are regarded as purely motor, the last are the supposed sensitive nerves of the muscle. Both kinds of nerves result from the subdivision of dark-bordered fibres which enter into the formation of the large bundle which is distributed to the muscle. But the so-called sensitive fibres may be traced a very long way from the large nerve trunk, and many of their finest subdivisions, of $\frac{1}{5000}$ of an inch in diameter, or less, may be seen to run on in company with other nerve branches, and can be followed into trunks composed of fine fibres, which pass away from the muscle at different points of its borders, some passing to the cutis, some to the connective tissue with which the muscle is continuous at the inner and outer edge, and others issuing from the muscle near its insertion into the fibrous tissue which gives origin to the abdominal muscles.

But some of the so-called sensitive-fibres are seen to pursue a still longer course. Even from these, branches are given off which may be followed to muscular fibres, and may be seen to divide upon the surface of the sarcolemma in precisely the same manner as is observed with regard to dark-bordered fibres which result from the immediate division of the large fibres constituting the original bundle (Plate XI., fig. 3).

The general course and distribution of the nerves to the pectoral of the frog are the same in the young, fully-developed, and old frog, except that as age advances, the fibres which are followed to, and seen to be distributed in, the connective tissue covering the muscle increase in number. In very young frogs, as far as I am able to observe, all the fibres resulting from the division of the primary fibres ramify upon elementary muscular fibres, or are to be traced to the vessels of the muscle. At this period the thick layer of connective tissue present in the adult is so thin that only indications of it can be discovered.

All these so-called sensitive fibres cannot be traced into, or shown to result from, the division of one or two of the dark-bordered fibres of which the bundle is constituted, as stated by Kölliker. So that there is nothing in the arrangement, course, or destination of these fibres to lead us to consider them sensitive fibres, and until new facts are brought forward in

favour of the opinion entertained by Reichert and Kölliker, it can scarcely be accepted.

1. It is certain that some of the branches of the supposed sensitive fibres of the trunk are lost on the sarcolemma and are distributed in precisely the same way as the fibres regarded as purely motor.

2. That the branches from which nerves distributed to the sarcolemma are derived, also give off fibres which are lost in the intermuscular connective tissue or in that upon the surface of the muscle—that is, the fibres considered to be purely motor give off some of the branches regarded as purely sensitive. And hence it results that all the branches considered by Reichert and Kölliker to be purely motor are not so, nor are all the fibres regarded by them as sensitive of this nature.

It remains to be proved if any of the fibres ramifying in the connective tissue upon and amongst the muscular fibres are sensitive. My own opinion is that there are no special sensitive fibres distributed to the muscle, and that the fibres in the fully-formed muscle considered to be of this nature are motor fibres, some of them probably having supplied at an earlier period of life muscular fibres, which have since disappeared, and these nerve-fibres now seem to be distributed to the connective tissue covering the muscle. I think it probable that the only fibres which have the power of transmitting sensations from the muscle to the nervous centres are those which are distributed to the capillary vessels, or some of those filaments which leave the muscle and pass into the bundles of cutaneous nerve-fibres.

Of the "Nerve-tufts" of the Breast Muscle of the Frog.

The question concerning the arrangement of the nerve-fibres in voluntary muscle receives elucidation from a study of the nerves distributed to the so-called 'nerve-tufts' of the breast muscle of the frog and figured by Kölliker (Proceedings of the Royal Society, May 1st, 1862, p. 78). This figure I have had copied (see Plate XVI, fig. 1). It does not, however, represent all that is to be seen in these swellings, for the numerous oval nuclei, figured in Kölliker's drawing, are represented by him as being pretty generally diffused throughout the swelling, and as not connected with one another or with any definite structure, nor is the relation of the nerve-fibres to these nuclei indicated by Kölliker.

Some of these nuclei are, however, seen in my specimens to be nuclei in the course of very fine nerve-fibres—nuclei which

take part in the formation of the nerve-fibres themselves. Others are the nuclei of the muscular fibres which are undergoing development, over the surface of which the fine nerve-fibres are spread out. These facts are demonstrated in several specimens which I have mounted in strong glycerine and acetic acid syrup.* A portion of one of these is represented in Plate XVI, fig. 2. This drawing which is magnified 700 diameters appears somewhat confused owing to the very close proximity of the nerves and muscles. It is, however, a careful copy of one of my specimens just at the spot where three dark-bordered nerve-fibres pass into or emerge from one of the 'nerve tufts.' The 'explanation' of this figure should be referred to. In fig. 3 I have shown one branch of a dark-bordered nerve-fibre and its division into the very fine fibres. These may be followed for a considerable distance amongst the developing muscular fibres.

It has been truly stated by Kölliker that the apparently single muscular fibre bearing the swelling is really a bundle of very fine fibres, varying from three to seven, or more, in number, and that the apparently p e n e t r a t i n g nerve-fibres merely pass between these imperfectly developed muscular fibres. I cannot however agree with him in the view that the fine muscular fibres result from longitudinal splitting of the fibres. The bundles of fine muscular fibres under consideration, extend at a certain period of their development from one extremity of the muscle to the other, but all the muscular fibres of the bundle do not reach so far. In one bundle sometimes ten or twelve distinct muscular fibres very closely packed together may be counted. Near the swelling the muscular fibres are wide, and many often terminate in fine pointed extremities. In fact at this point several spindle-shaped, nucleated, transversely striated muscular fibres may often be seen, and the stages through which the elementary fibres of voluntary muscle pass in their development may be traced.

In these "nerve tufts" we may indeed study in the fully-formed animal, striped muscle and nerve in every stage of development. Vessels cannot be traced into the youngest tufts, but in those which consist of several partly grown muscular fibres capillaries are to be seen, so that the development of muscles, nerves, and vessels can be studied in these imperfectly developed "tufts."

From the above observation, it will be seen that I cannot

* I have found this strong acetic acid syrup a most valuable agent in these and kindred investigations.—"How to Work with the Microscope," third edition, p. 202.

agree with Kölliker in the view he has taken of these bodies. He says, "Now if it be admitted that the finer muscular fibres composing the bundle are generated by the division of thicker muscular fibres, as Weismann justly concludes, the explanation of the nerve tufts becomes easy, inasmuch as they may be conceived to arise from a simultaneous growth and division of the nerve fibre belonging to the parent muscular fibre, in order that each of the young muscular fibres may obtain its branch of nerve" (Croonian Lecture, May, 1862).

Kölliker thinks that the narrow young muscular fibres result from the division of old ones. So far from this being the case, the young muscles and young nerves are developed from collections of nuclei or masses of germinal matter, precisely resembling those which are found in the embryo. If new muscular fibres are to be developed from old ones, the 'nuclei' increase in number, the old muscular tissue undergoes disintegration, and is absorbed, and a mass consisting entirely of nuclei, or masses of germinal matter, by the agency of which the formed material of the new fibres is at length produced, results.

As I have pointed out on many occasions, in fully formed organs there exists a certain proportion of embryonic germinal matter, which undergoes development at a future period of life, and if this becomes fully formed tissue, still there remains embryonic matter for development at a still later period, and so on. In the situations of these so-called nerve tufts in the breast-muscle of the frog, new elementary muscular fibres are added to those already formed, and the muscle grows as the frog advances in age. And in the formation and growth of the muscular fibres, and in the formation and arrangement of the nerves around them, the movements of the several nuclei or masses of germinal matter to which I have drawn attention, play no unimportant part. (See my paper on 'The movements of the living or germinal matter of the tissues of man and the higher animals.' Archives, Vol. IV., page 150).

With reference to the nerves supplying these so-called nerve tufts, I would remark:—

1. That two dark-bordered nerve-fibres, running in the same sheath, may often be traced to one part of the 'nerve-tuft.'

2. Besides the dark-bordered fibre or fibres, there are invariably very fine fibres running in the same sheath.

3. That the dark-bordered fibres and the accompanying fine fibres divide and subdivide very freely amongst the young muscular fibres, and that thus quite a leash of very fine nerve-fibres results, in the course of which numerous nuclei exist at certain intervals. Many of these can be followed upon or between the

muscular fibres, for the distance of the twentieth of an inch or more from the oval swelling (see fig. 5, Plate XIII).

4. That the dark-bordered fibre or fibres which enter at the tuft are not the only nerve-fibres distributed to these bundles of muscular fibres, but that invariably a bundle, consisting of two or three fine, but dark-bordered fibres, is connected with the muscular fibres, at a point above or below that at which a swelling is situated, where the large fibre or fibres enter. Sometimes there are two such bundles, one above and one below. These not unfrequently give off branches, just before they pass to the muscular bundle, which pursue a longer course, and are distributed to other larger muscular fibres, and oftentimes branches pass from one muscular bundle to more distant ones.

From the above observations it follows, that these 'nerve tufts' in the breast muscle of the frog, consist of developing muscular fibres, which are freely supplied with nerves, and that the number and distribution of the nerves render it probable, not only that there are entering and emerging fibres, nerve loops, and plexuses, or networks upon the muscular fibres, rather than free ends, but that the contraction of the new muscular fibres are harmonised with those of the other and older elementary muscular fibres of the muscle by commissural branches of nerve fibres.

I think it not improbable that in some cases the organs termed 'end plates' are of the nature of these so-called nerve tufts in the breast muscle of the frog, that is, that so far from being special organs in connection with one individual muscular fibre upon which they are found, they consist of masses of imperfectly developed structure—of tissues in an embryonic state, which would have undergone development into fully formed nerves and muscles at a future period of life. Certain it is, that all these 'end plates' are external to the sarcolemma; that into every one that I have seen more than one nerve-fibre, and oftentimes as many as six or eight may be traced, and that they are never placed, as has been represented, at the end of a long branch of nerve-fibre, but are always appended as it were, to a nerve trunk, crossing numerous muscular fibres, and invariably composed of more than two fine nerve-fibres. Oftentimes nerve-fibres may be seen passing away from the 'end organ.' I do not, however, mean to imply that these apparent end organs are all of this nature. Some undoubtedly very closely resemble in their arrangement some of the nerves upon the elementary fibres of the breast muscle of the frog, and like these lie upon the sarcolemma, and are adherent to it. Into every one that I have seen, more than one nerve-fibre passes. These 'end organs'

are very distinct in the muscles of many lizards, and especially in the cameleon; I propose to publish drawings of the 'end organs' of the latter animal shortly. Kühne's latest figure of these end organs has been copied from his paper published in the 30th volume of Virchow's Archiv., 1864, and is represented in Plate XV., fig. 3, reduced to half its size. Kühne asserts that the structure represented lies beneath the sarcolemma, and one terminal fibre passes into it. As I have recently prepared some very good specimens of these bodies, I propose to discuss the question fully in a separate memoir.

Further Observations on the Nerven-hügel.

In the Comptes Rendus for November 21, 1864, M. Rouget endeavours to show that Kühne's Doyèreschen Nerven-hügel in the muscles of certain invertebrata result from the sarcolemma being drawn up in a cone, as the nerve which adheres to it is stretched, and states that, at the nerven-hügel, the nerve-fibre divides into two fine fibres, which may be traced for some distance, and then terminate. "Leur extrémité terminale est légèrement effilée; elle ne présente ni plaques, ni noyaux, ni substance finement granuleuse."

The structure of these nerven-hügel in insect muscles was fully described by me in a paper, accompanied by several drawings, read to the Microscopical Society on June 8th, 1864, and published in the 'Transactions' on October 1st, 1864, or three weeks before the appearance of M. Rouget's memoir. Rouget confirms my statements with reference to the production of the nerven-hügel, but we are at variance upon the further progress and mode of termination of the nerve-fibre, Rouget maintaining that it penetrates beneath the sarcolemma and terminates in a very fine fibre, in contact with a very limited portion of the contractile tissue, while I have been able to trace the nerve for a long distance beyond the point at which he makes it end, dividing into very fine fibres, which form an extended network upon the sarcolemma. Rouget's researches, however, as far as they have gone lead him to conclude that the arrangement of the nerves in the muscles of articulata is totally distinct from that met with in vertebrata. "Il résult de ces faits qu'il n'y a pas identité entre les divers modes de terminaison des fibres nerveuses motrices chez les vertébrés et les articulés." On the other hand my observations lead me to the conclusion that the arrangement is in its essential points the same in all. In no case are there nerve-ends, but always plexuses, or networks,

which are not in structural continuity with the contractile tissue of muscle. In no case is the distribution of the nerve-fibre confined to a very limited extent of muscle as is maintained by most continental anatomists.

I trust that in future all writers upon this question will give drawings of the appearances they have observed, for mere description without drawings can do nothing towards clearing up the many conflicting statements.

The Demonstration of the actual Arrangement of the very fine Nerve-fibres upon the Sarcolemma of the Elementary Muscular Fibres.

I have already described the arrangement of the finest nerve-fibres in the mylohyoid of the hyla, and have pointed out that in this muscle fine fibres may be actually traced from one undoubted nerve-trunk to another (Proceedings of the Royal Society, June 5th, 1863). The plate illustrating this paper is reprinted in Plate XII.

I have recently succeeded in preparing a specimen of the breast-muscle of the frog, in which the arrangement I had inferred must exist from the facts recorded in my earlier papers, is actually demonstrated, and the fine fibres resulting from the division of the dark-bordered fibres, and the fine fibres running in the sheath become continuous with a network of very fine fibres ramifying upon the surface of the sarcolemma, and, as it were, beneath the connective tissue. At intervals nuclei are seen as in other cases. In fact this plexus or network upon the surface of the sarcolemma of the muscles of the frog very closely resembles, in its general arrangement, the network of nerve-fibres in the heart, tongue, bladder, cornea, and connective tissue of the same animal. Its extreme delicacy renders it very liable to destruction, and as the thickness of the elementary muscular fibres, and the numerous lines existing in their structure interfere with the passage of the light, it is not surprising that there should be great difficulty in demonstrating it.

The nerve-fibres constituting this plexus, although compound, are so fine that they are quite invisible unless very high powers be employed, and the specimens specially prepared. In recent specimens of muscle not the slightest indication of the extensive system of fine fibres which I have described is to be seen, nor is the muscle peculiar in this respect, for the finest nerve-fibres cannot be seen in recent specimens of the frog's bladder, pericardium, connective tissue, &c. So pale and

delicate are these fine nerve-fibres that they cannot be seen even in the most delicate connective tissue until the latter has been rendered transparent, and the nerve-fibres themselves granular. The nuclei in connection with them are lost amongst the numerous nuclei of the connective tissue, in which the nerve-fibres lie. By the slow action of acetic acid upon specimens immersed in glycerine or syrup, the fine nerve-fibres are decomposed and rendered granular, in consequence of minute oil globules being set free. This change has enabled me to trace the course and distribution of the finest nerve-fibres in a great many different tissues.

By proceeding as I have elsewhere fully described, the appearances to which I have drawn attention may be demonstrated.*

I claim, therefore, to have demonstrated that nerve-fibres passing to a muscle divide at length into a vast number of exceedingly fine pale granular fibres, which ramify upon the external surface of the sarcolemma, connected with which fibres at certain intervals, are oval nuclei (Phil. Trans., 1860), and that these fine fibres after an extensive and in many cases very circuitous course, join with other fibres to form dark-bordered fibres, which at length pass towards the nervous centre either in the same bundle as the dark-bordered fibres passing towards the muscle or in other bundles.

It therefore follows, that of the dark-bordered nerve-fibres distributed to a muscle, some pass from the nervous centre towards the muscle, some from the muscle towards the nervous centre, and that the nerve-fibres do not end in the muscle at all. The results of these researches, considered in connection with those arrived at from the investigation of various kinds of nerve centres, justify the inference that the fundamental arrangement of a nervous apparatus is a complete and continuous circuit.

My position in this enquiry is as follows:—

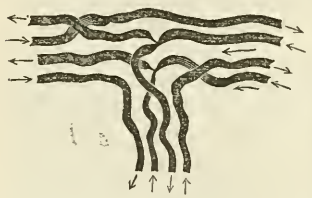
1. I was led to the conclusion of the existence of a network of nucleated nerve-fibres, many, if not all, composed of finer fibres upon the surface of the sarcolemma of the muscles of the mouse in 1859, the observations being published in the Phil. Trans. for 1860.

2. That after having studied the conclusions arrived at by Kühne and Kölliker upon the breast muscle of the frog, and those of other observers opposed to my own views, I have, after working for a very long time upon the same muscles employed

* How to work with the Microscope. 3rd Edition, 1865.

by them, succeeded in tracing the nerve-fibres a much greater distance from the dark-bordered fibres, and have demonstrated that certain fine fibres ramifying in the sheath of the nerve are not connective tissue, as usually stated, but true nerve-fibres, which are nearer to their ultimate ramification than the dark-bordered fibres with which they ramify.

3. That the arrangement of the fine nerve-fibres in the form of networks or plexuses upon the surface of the muscle, beneath the skin, and in other parts, resembles that of the dark-bordered fibres in the same situations which has long been known. In fact the networks and plexuses of fine pale nucleated nerve-fibres in the embryo which become the networks and plexuses of the dark-bordered fibres of the fully developed animal, resemble in all particulars the networks and plexuses of fine fibres described by me in the latter, and that in all cases where a trunk composed of fine fibres or coarse fibres branches off from another trunk, the former invariably consists of fibres pursuing opposite directions, as shown in the figure.



4. That by independent observations upon the bladder, small arteries, the skin, connective tissue, cornea, papillæ of the tongue, and other tissues of the frog, I have proved that networks, the fine fibres entering into the formation of which are compound, or plexuses of very fine nerve-fibres, exist, and that nerves do not terminate by free extremities in any of these tissues.

5. That a number of observations I have made upon the minute arrangement of nerve-fibres in the trunks of nerves and upon the origin of nerves from nervous centres in numerous vertebrate and invertebrate animals, favour the conclusion that in all cases complete nerve circuits exist, while during many years of careful observation I have not been able to discover one fact which is incompatible with such a view.

6. That by direct observation upon the thin mylohyoid muscle of the frog, finer fibres than have been hitherto described have been demonstrated, and that some of these have been followed from one compound nerve-trunk over a great many elementary fibres to another trunk. Many of the nerve-fibres described by me in my recent memoirs are so very fine, that it is not surprising that they should have been regarded by many observers as delicate fibres of fibrous tissue. In my preparations their true nature is however demonstrated by the change

induced by the action of acetic acid, and by their continuity with true dark-bordered nerve-fibres.

7. That in the auricle of the frog's heart and also amongst the elementary muscular fibres of the frog's tongue very fine nerve-fibres, not more than the 1-60,000th of an inch in diameter, have been traced from one nerve-trunk to another.

8. That similar facts have been observed in the breast-muscle of the frog, and fine fibres have been traced from one trunk to another.

9. That from all this evidence the conclusion is, that nerve-fibres never end or terminate by free extremities, but that in all cases complete circuits exist, and the inference is justifiable that a circuit is the fundamental arrangement of a nervous apparatus.

A CONTRIBUTION TO THE ETIOLOGY OF TYPHOID FEVER— A RECENT OUTBREAK AT CHARMOUTH, DORSETSHIRE.

By W. B. Kesteven, F.R.C.S.

The village of Charmouth, about two miles to the east of Lyme Regis, consists of a single street running nearly east and west up a steep hill, parallel with the coast line. It is ordinarily one of the healthiest villages in England, enjoying the advantages of an abundant water supply, and the purest of air, both from the sea and beautiful country inland. The houses are, for the most part, detached, or semi-detached villas and cottages, on each side of the main street, which has neither back streets nor crowded courts running off from it. At the foot of a hill a small river, celebrated for its trout fishing, finds its way through pleasant meadows to the sea.

Charmouth is situated on the Lias beds, which offer great attractions to geologists. These strata dip towards the coast at a moderate angle to the horizon and form usually a reservoir, whence issues through countless land-springs an abundant supply of clear and wholesome water.

Thus, under ordinary meteorological conditions, this village possesses all the natural advantages essential to public health, and thus its reputation in all sanitary respects has stood deservedly high. These natural advantages had been still further enhanced by the construction, a few years back, of a sewer down the street, emptying its contents into the stream that passes beneath a bridge at the foot of the village, towards the sea.

During the last summer, however, the long continued

drought had changed this favourable state of things, assisted, as it seems to me, by one fatal oversight on the part of the inhabitants of Charmouth. It has been stated that water flows abundantly by land-springs from the surface of the soil. These may be seen in many of the gardens in front of the cottages that line the road side. The water that is not used is allowed to run to waste by any channels it may find into the fields or brooks. Having observed the early course taken by the fever in this village, I am disposed to attribute its first appearance entirely to sewerage emanations arising out of this waste of water that should be used for flushing. The first cases occurred at the lowest level, the disease gradually creeping up the hill. This would be the result of the retarded flow of the drainage, and consequent accumulation of sewerage at the lower end of the drain. I had taken a house at this part of Charmouth for six weeks, on account of previous ill health in my family. On the day before our return to London one of my children, a boy of eleven years of age, was ill, and in the course of two or three days afterwards two younger children followed suit. All had enteric fever, with the well-marked typhoid eruption; all suffered severely and were not convalescent in less than six or seven weeks. No other cases occurred in our household after our return; but I am informed by my friend Mr. Norris, of Charmouth, that he has had many cases of typhoid since; that four cases proved fatal; and that in almost every case the disease could be traced to bad drainage.

There existed, however, in Charmouth, as in most other country villages, a further possible mode of the propagation of the fever, which would explain its occurrence in several members of one household, more closely than by supposing contagion, the communication by which is not the characteristic of typhoid, as it is of typhus. The water supply of most of the houses is got from wells in the gardens, which, owing to the geological structure of the country, need not to be dug deep before, in ordinary seasons, a copious flow of water follows. During the past autumn these wells were, many of them, dried up, or ran very short of water. Not far from the wells there would be found, in most instances, a privy with its cesspool. It is clear that with such a geological structure as exists in Lias strata the diminution of the water from the wells would induce a percolation of the fluid contents of the adjacent cesspools. This would not be indicated either in the taste or smell of the drinking water, as it is well known that water loaded with the deleterious nitrates, under such circumstances, has rather a pleasant flavour.

I believe that such was the mode of conveyance of the poison, in some cases, but it should be added that when the water in the wells became very scarce, the cottagers at the lowest part of the village were driven to take their supply from the river, into which the drain emptied itself, and thus, furthermore, the disease may have had its origin in some instances.

In the history of this outbreak we have additional evidence that typhoid fever may be developed, from the emanations of sewers. These conditions, it is true, being, in the instance now related, beyond the control of man, viz., the excessive heat and long drought; although it remains very probable that had the means so abundantly at hand been made use of for flushing the drains, the conditions which gave rise to the epidemic would not have been produced. Had the surplus water been conducted by a few pipes into the sewer, the waste water would have done good service in flushing the drains, and probably would have averted an outbreak of typhoid that occurred during the past autumn, the village having been previously free, and, indeed, generally exempt from fever. If this view be correct it follows that the inhabitants of Charmouth have in their own hands the power to prevent the recurrence of such an outbreak in their delightful and otherwise most salubrious village.

This circumscribed outbreak bears out exactly the received doctrines on the non-contagious nature of enteric fever. No case had occurred in this village for upwards of seven years, and on that occasion the disease had been traced to defective drainage. The cases that occurred were all typhoid, or enteric, they were met with in the better class of houses as frequently as among the poor; in short this outbreak was but a repetition of what has been recorded of other limited epidemics, that where more than one person in a house were attacked, all had been alike exposed to the sewage emanations; that it was not traceable in any degree to overcrowding, and that if transmitted by other means than the poisoned atmosphere from the drains, it was conveyed by the contamination of the water supply from the infiltration of sewage from the previously infected.

ON THE OCCURRENCE, DIFFUSION, AND ACTION OF THE CONSTITUENTS OF THE BILE IN THE ANIMAL AND VEGETABLE ORGANISM.

By Dr. F. W. Beneke, Acad. Teacher in Marburg, Giessen, 1862.

(Abstract by Dr. Duffin, Asst. Physician, King's College Hospital.)

In the sixth volume of his Archives (1854), Virchow published a paper "On the Extended Distribution of a Substance Analogous to Animal Nervous Matter." This substance he named myelin (markstoff), suggesting that even should it not ultimately prove a simple body, its occurrence in a uniform thoroughly homogeneous state as much vindicated its claim to a distinctive title as is the case with albumen, syntonin, fibrin, etc., which are also probably compounds.

Virchow describes this substance as characterised by its peculiar brilliancy, and by the strange forms that it assumes. "At one time I saw long threads resembling large primitive nerve-fibres in breadth and shape, extending across the field of vision. They generally exhibited a fine clear axis, readily comparable to an axis cylinder, and a broad, double, definite outline, the outer of which was darker than the inner. They either terminated in a rounded definite margin, or they formed a knot of closely coiled contorted bands, often of irregular thickness, from which again large drops with a double outline sometimes issued. In other situations large, rounded, concentrically striated bodies appeared, suggesting that one of the threads just described had been coiled round itself. This supposition was the more likely, as often processes shot out from these coils that were themselves again coiled at their extremities. At other points the processes ended in small rounded or oval drops, still retaining the double outline. Other masses presented a dull, homogeneous, or partially plaited aspect, round the margin of which the double line everywhere was to be traced. From these double contours, varicose threads shot out in many directions, ending in the most variously twisted and knotted masses. Then some curious things were seen, resembling rolled up pieces of paper, doubly contoured, and included in crooked, somewhat undulating lines. Lastly, smaller forms were very frequent, simple, not doubly contoured, pale, resplendent drops, of the size of a blood corpuscle and less; longer, doubly contoured drops, either spherical or prolonged into a small, short thread;

smaller, shorter threads, with a double outline and perfect termination, often with a doubly contoured drop resting upon them, so as to closely resemble fungi. All these formations consisted of a viscid material, the flowing of which could be often observed as the long, nerve-like threads issued from the general more coherent mass."

Virchow first obtained this substance from diseased pulmonary tissue, but he shortly afterwards procured it from the gall-bladder, a hepatic cyst and the ovary of the calf. He was next able to verify its abundant existence in every spleen, and he also extracted it from the thyroid, from the medullary part of the brain, from fresh pus, and from fibrin. He filtered a hot alcoholic extract, and brought the pellicle which formed on the filtrate with some fresh alcohol into the field of the microscope. Experience seems to leave little doubt that it occurs in the interior of the tissues themselves, and as we may conclude that it is constantly present in small quantities, even in the blood itself, it would not be too much to assume that it is one of the most diffused principles of the animal economy. Of its chemical properties Virchow thus writes, "Easily soluble in hot alcohol, a part only deposits on cooling. In water the mass swells up to an extraordinary degree. It is when thus swollen that it presents its morphological peculiarities. Ether, chloroform, and turpentine readily dissolve it; weak acids and alkalis have little influence; strong alkalis cause it to shrink; strong acids make it swell still further, and then destroy it; chromic acid renders it hard yellow; strong sulphuric acid colours it red, occasionally violet." (Op. cit. p. 566.)

From the time of the publication of Virchow's paper, myelin seems to have been lost sight of, till Mettenheim found it in the human lens. The only addition that he, however, made to our acquaintance with it was the discovery of its polarising properties whilst in alcoholic solution, properties of which water deprived it. The earlier notices as to the nature of myelin are peculiarly scanty. Reasoning from some ultimate analyses, Virchow observes, "From its proportion of nitrogen and phosphorus, from its swelling in water, its behaviour with potash, its combination with lime, we are strikingly reminded of Frémy's cerebric, and oleophosphoric acids." Kölliker* also discovered a substance analogous to these same acids in the semen of the carp, and was struck with its resemblance to the myelin of Virchow, but did not pursue the subject. Little indeed seems to have been done to establish either the chemical constitution

* Zeitschrift für wissenschaftl: Zoologie, p. 257.

or the physiological properties of this substance till Prof. Beneke was gradually led to adopt the views it is the object of this paper to enunciate.

Whilst experimenting with concentrated sulphuric acid on a variety of tissues, he noticed the coloration taken up by the nuclei of some and the fluids surrounding others to be reddish, yellowish, or violet; thus the nuclei of the cornea and the fluid surrounding the lens of the calf acquired the latter tint, whilst the epithelium of the skin of the fœtus underwent a regular play of colours, yellow, reddish, carmine. With an epithelial cancer the play of colours was so marked as to suggest the presumption of nerve fats, and the possibility of cholesterine or myelin, and Beneke particularly noticed the coloration to be most intense about isolated fat drops. The surrounding fluid of the preparations also gradually got tinted, suggesting that the body causing the colour was soluble in sulphuric acid. This last fact led Beneke to suspect the presence of the biliary acids. He was thus induced to test microscopical preparations with a sugar solution (1 sugar to 10 water, in the later experiments 1 sugar to 4 water), followed by a drop of concentrated sulphuric acid. He first tried a cancroid of the penis. A play of colours ensued through yellow, red, carmine to violet, and was most intense at the nests of the cancroid, at the large nuclei of the cancroid cells, and on a few isolated fatty particles. He then exposed with very similar results the tendo achillis of the calf, the cornea of the calf, and permanent cartilage. The cartilage cells especially acquired a deep coloration. The lacunæ of bone became deeply tinted. In from five to ten minutes fat globules would assume a deep rose or violet hue, etc. The presumption was thus raised that all the tissues contained a material yielding to Pettenkofer's test the reaction of the biliary acids. Struck with the vividness of the tints afforded by both normal and diseased epithelial structures in rapid growth, in multiplying cartilage cells, and in the nervous centres, he concluded that the material in question was of great importance in cell formation. This led him to prove the behaviour of yolk of egg, where he found the reaction in great splendour. Immediately on the addition of the reagent a red colour passing into violet was developed, and at the end of six hours this had become deep violet.

These observations he supplemented by the inquiry, do only the biliary acids, or do other constituents of the animal organism yield a reaction with sulphuric acid and sugar, such as Pettenkofer has described for the former?*

* The following are Pettenkofer's directions :—" Pour a portion of the sus-

Butyric, baldrianic, caproic, caprylic, and caprinic acids all gave positive results to the colour test, but never with great intensity. The reaction of lactic acid was so indefinite, that it may practically be considered as insensible to the test. Oleic acid on the contrary gave the nearest approach to choleic acid of any animal substance. Stearic acid and stearin gave very imperfect results, margaric acid and margarin gave none. The conjugate acids glycocholic and taurocholic behave just like their non-nitrogenous constituent cholic acid. This and not the nitrogen holding glycin, or the sulphur holding taurin gives the reaction. Choloidic acid behaves similarly. The reaction of cholesterine varies with the degree of concentration of the sulphuric acid. With certain dilutions a play of colours is indeed obtained, but this is always very transient, and passes into a dirty green. Under the microscope the edges of the crystals become successively yellow, yellowish brown, yellowish red. At last the crystals melt away, and come to resemble a fluid fat. All the ordinary animal albumens must be thoroughly purified from fat, but pulverized lens, the albumen of bullock's blood, and fibrin nevertheless exhibited Pettenkofer's reaction, although less forcibly than before the extraction of the fats. This led Beneke to ask whether the albuminates may not be considered conjugate substances, one of the factors being non-nitrogenised, and acting the same part as the cholic acid of the tauro and glycocholic acids? The idea is supported by the absence of even a trace of reaction between sugar and sulphuric acid, and all exactly known nitrogenised bodies, as leucin, tyrosin, taurin,

pected fluid into a test tube, and add English sulphuric acid, guttatim, to about $\frac{2}{3}$ the volume of the fluid, whereby the temperature is considerably raised. The addition must be made so gradually that the temperature shall at no time exceed 50° R., as otherwise the choleic acid is too much changed. Then add 2—5 drops of ordinary cane sugar solution, containing 1 part sugar to 4—5 parts of water, and shake the whole. If choleic acid be present, a more or less deep violet red colour will be produced, according to the amount of bile present."

Neukomm (ueber die Nachweisung der Gallensäuren, &c., 1860) proposes the following modification (p. 107):—"A single drop of a $\frac{1}{10}$ per cent. solution of cholic or glycocholic acid will yield a splendid purple violet colour if it is brought in contact with a drop of dilute sulphuric acid (4 water 1 sulphuric acid) and a trace of sugar solution in a porcelain cup, and then gently warmed over a spirit lamp. As 1 cubic centimetre equals about 8 drops, it is thus possible to demonstrate $\frac{6}{1000}$ milligr. of biliary acid with complete accuracy." As a further test, he suggests (p. 117) "The biliary acid or salt is to be sprinkled with a small quantity of concentrated sulphuric acid moderately warmed, and then water added. The resinous flocculi that subside are to be separated from the acid, washed with water, but not so as to remove all the sulphuric acid, and then again gently heated in a porcelain cup till coloration ensues. If the residue be taken up in a small quantity of alcohol, and the green solution be evaporated, the interior of the cup will be coated with a deep indigo blue film, even when but little acid has been used. If the biliary acids have been impure, or the sulphuric acid or the temperature react too long, the pigment film will be green."

urea, etc. Hence all the fatty acids of the body, but particularly the oleic, react in some measure to Pettenkofer's test. The albuminates do the same, and the more markedly, the less completely they have been freed from fats. This test consequently does not enable us to draw any absolute conclusion as to the presence of a biliary acid; but inasmuch as the proper play of colours with the biliary acids is peculiarly characterised by clearness and intensity, and the purple-violet tint is retained for a considerable time, these signs are strongly suggestive of the presence of cholic acid or its compounds, and the test is still possessed of great histo-chemical value.

These results led Beneke to apply Pettenkofer's test to the alcoholic, ethereal and chloroform extracts of a variety of tissues. An alcoholic extract of the yolk of egg gave results as splendid as those obtained from pure cholic acid. But the oleic and other fatty acids which are contained in the yolk of egg are with difficulty taken up by alcohol, albuminous compounds even less; thus the idea of the biliary acids or of their derivatives was again suggested. The alcoholic extract of a number of other tissues, *e.g.*, cartilage, muscle, brain, lens, also gave more or less marked results to sugar and sulphuric acid. The ethereal extracts required to be first dried and taken up by alcohol, as the ether itself, however pure, tends to give the reaction in question, but with this precaution the results were the same as above. The chloroform extracts also gave the characteristic play of colours, but much less vividly. The extracted albuminous tissues, as might have been anticipated, continued to answer to the test. It matters not, however, how in the latter case the reaction is set up; it is certainly not produced by an albuminate in the former, whilst its intensity suggests the presence of biliary acids.

The other properties of the cold alcoholic extracts of animal tissues proved also very instructive. They had all a more or less fatty character, and eagerly absorbed water. All burning with a distinct acrolein odour, they, for the most part, emitted no smell of burning nitrogenised matter, and left a small phosphatic residue. Some of the residues, particularly those from cerebral tissue and yolk of egg, contained crystals of cholesterine. All, when treated with water, swelled up to form a tough, viscid mass. With excess of water a great part of the majority of them dissolved; sometimes the whole. When this process of tumefaction was watched under the microscope, the forms ascribed by Virchow to myelin were severally observed—gradually vanishing as the substance dissolved in the water. Weak alkalis assisted the development of the forms, whilst the

acetic and mineral acids checked it; concentrated sulphuric acid rapidly dissolved them, at the same time inducing a faint violet coloration. These results were obtained with alcoholic extracts of the contents of the jejunum of a dog that had been fed five hours previously on fat beef, from the chyleducts of the mesentery of the same dog, as also from its spleen, venous, and arterial blood. He also got them with the purest fibrin that can be obtained, and from dried blood albumen, from yolk of egg, and from a long list of healthy and diseased structures.

Proceeding to a closer analysis of the myelin thus frequently obtained, Beneke started from the distinct acrolein smell it emitted on burning, the intense reaction of biliary acids it evinced to sugar and sulphuric acid, and its solubility in water. This last peculiarity was strongly opposed to its being any ordinary fat, whilst the first was equally presumptive of the presence of one or more glycerine fats, or of the glycerine phosphoric acid discovered by Gobley in the yolk of egg. Beneke employed the yolk of hen's eggs in his further investigations. He saponified with potash, and boiled the mixture from 1½–18 hours. A clear, golden yellow viscid mass resulted on cooling, in itself almost conclusive of the presence of a glycerine fat. On decomposing with hydrochloric acid, and collecting the fatty acids on a filter, the evaporated filtrate developed when burnt the acrolein odour; but this also occurred with the mass on the filter, which, moreover, yielded the biliary acid re-action more brilliantly than before saponification.

After the mass on the filter had been taken up by hot alcohol, and the latter had been expelled, the residue proved very hygrometric, and developed the myelin forms on the addition of a potash solution. If the alcohol were slowly evaporated, crystals developing the myelin forms first appeared; then crystals of margaric acid; lastly, a volatile fatty acid, probably oleic.

Did the mixture of substances composing the myelin of Virchow contain nitrogen? As already observed, the alcoholic extracts of brain, egg yolk, etc., when incinerated, emitted an odour of burning albuminates. Frémy had already produced a nitrogenized acid, the cerebric, from brain extracts, and Beneke is now prepared to assert the presence of nitrogen in the extracts of egg yolk and of muscle, but the nitrogenized constituents are in extremely small quantity.

The further processes were conducted as follows:—The alcoholic extracts of a number of yolks were dissolved in water, and converted into a lead soap by means of litharge. After decantation the lead precipitate was extracted with hot alcohol,

filtered, and the extract cleared of lead by sulphuretted hydrogen. On cooling, a myelin free of any of the ordinary saponifiable fats was obtained. The shapes of these crystals resembled those ascribed by Virchow to a special form of cholesterine, but they yielded the cholic acid reaction perfectly, and their great solubility in water was opposed to their being cholesterine; still cholesterine they proved to contain. As this substance is almost insoluble in cold alcohol, this was used to wash the mass, which was then recrystallised out of hot alcohol as snow white silky crystals. These, on ultimate analysis, gave the following results:—

HO	= 0·2195	= 11·98 H*
CO_2	= 0·6255	= 84·01 C
O		= 4·01 O
		100·

But what was the residual substance that imported solubility in water to cholesterine and caused it to assume the shapes of myelin? The intense reaction with sugar and sulphuric acid pointed strongly to the biliary acids. A myelin yielding extract was examined for chromogens according to the methods of Frerichs and Staedeler (see ante, note, p. 195). The honey-like, golden yellow extract was dissolved in concentrated sulphuric acid. A viscid solution, with a carmine red edge, was thus obtained. On the addition of cold water, the reddish tint vanished, and greyish flocculi fell. These being dissolved in fresh alcohol yielded to sugar and sulphuric acid a distinct reaction of cholic acid. When these flocculi were warmed with a little spirit in a porcelain cup, the vessel became coated with a blue, slightly reddish covering. The next day this covering was green.

The alcoholic extracts of the lead soap were then evaporated to two-thirds their volume. Allowed to crystallize in the cold, they precipitated fresh quantities of cholesterine. These were filtered off, the filtrate again reduced, and crystallized out, and so forth, till only yellowish flocculi appeared in the deep golden yellow mother-liquor. Thus a mother-liquor was obtained, from which the greater portion of the cholesterine had been removed, but which must still contain any biliary acids present. This mother-liquor, when treated with sugar and sulphuric acid, yielded the most intense reaction of biliary acid, but on evaporation no crystalline residue was found, but a tough,

* Schwendler and Meissner give the percentage of cholesterine as C = 84·2, – H = 12, O – O = 3·8 – 100.

golden yellow, hygroscopic mass, containing scarcely a particle of crystalline cholesterine. The mass dissolved easily in water, but, curiously enough, when examined with the microscope, none of the characteristic beautiful shapes of myelin were perceived, but only large globular masses without the remotest resemblance to it; but the addition of a fresh portion of cholesterine at once reproduced the form. Hence the presence of cholesterine is absolutely necessary for the production of myelin. But what was the fatty viscid body, soluble in water that had been now quite deprived of cholesterine? It had a neutral reaction, and when burnt yielded a feeble but distinct odour of acrolein. The suspicion of the presence of an oxide of lipyl combined with a biliary acid was thus raised. Beneke first essayed to obtain the biliary acids by prolonged boiling with baryta. To deprive the mother-liquor of any trace of cholesterine, the alcohol was expelled, and the residue dissolved in water; the solution was then boiled for ten hours with baryta water; it then reddened and deposited a resinous precipitate. The liquor, after filtration, was exactly neutralized with sulphuric acid, again filtered, and evaporated. A tough, brownish residue was thus obtained, containing a number of crystalline needles. The whole residue showed very little solubility in alcohol, and gave so slight a reaction with sugar and sulphuric acid as to banish the idea of the presence of cholic acid, but the residue, for the most part, dissolved easily in water, and then alcohol precipitated a yellowish tough mass, whilst alcohol acidified with hydrochloric acid, threw down a scanty whitish precipitate, consisting of the aforesaid needles.

The baryta precipitate itself, on the contrary, continued to react vividly to sugar and sulphuric acid. After the withdrawal of the baryta by sulphuric acid, it deposited first a trace of cholesterine, and later a tough, yellow, fatty body, identical with that acquired by direct evaporation of the mother-liquor. The same gave the biliary acid reaction still most intensely, but it had quite lost its solubility in water. When mixed with cholesterine and brought into contact with water, it refused to assume the forms of myelin, but the addition of alcohol still produced them.

These results would be inexplicable but for the researches of Kolbe* into the behaviour of taurin and glycin. Boiling with baryta separates the biliary acids into cholic acid, and its nitrogenized conjugates taurin and glycin. Both of these latter are soluble in water, insoluble in alcohol. Whilst glycin can com-

* Ueber die chemische Constitution u. Künstliche Bildung des Taurin. Annal. d Chem. u. Pharm. cxxii., Hft. i, p. 33.

bine both with bases and acids, taurin has no basic properties, but can unite with bases. Further, taurin remains in solution in alcohol containing some alkali, but is readily deposited from acidified alcohol, whereas glycin is readily precipitated from its watery and alkaline solutions by alcohol, but in an acid form is readily kept dissolved by alcohol.

Assuming, then, that the fat-like soluble body of the mother-liquor contained taurochlorate and glycochlolate of lipyl, it is not improbable that by boiling with baryta taurin and glycin may be retained in solution as taurinbaryta and glycinbaryta. Now the baryta was removed by sulphuric acid. When the residual solution was alkalized and treated with excess of absolute alcohol, a tough brownish mass separated, consisting essentially of glycin; and on the alcohol being acidified, a light whitish crystalline precipitate fell, probably taurin.

Beneke concludes that the body which was insoluble in water was cholate of lipyl, as it continued to yield the biliary reaction, and on the addition of cholesterine and alcohol developed myelin forms, whereas the glyco- and taurocholates developed the same properties to water or thin syrup.

Thus to sum up. There are in the alcoholic extract of the lead soap, firstly, cholesterine. When this has been as far as possible crystallised out, an unsaponified fatty body remains in the mother-liquor, from which boiling with baryta extracts, taurin and glycin (?), but leaves an unsaponified fatty body, giving an intense biliary reaction to sugar and sulphuric acid, and which Beneke takes to be cholate of lipyl.

All attempts to trace the biliary acids in healthy blood and tissues have, as is well known, hitherto been in vain; yet since the researches of Bidder and Schmidt it is scarcely doubtful that they re-enter the blood either directly or with the chyle. Is it then very unlikely that the constituents of the bile combine with the food whilst in the intestine, but that this combination has hitherto eluded us in the blood and the tissues? From the first Beneke considered it probable that the oxide of lipyl, when separated from its fatty acids, might combine with the biliary acids, and so pass into the blood, and that possibly a combination of cholesterin with the fatty acids might take place. These presumptions are countenanced by the following considerations:—

I. Bernard has indicated the faculty possessed by the pancreatic juice of separating the oxide of lipyl from its fatty acids, and although Lenz* correctly remarks that the acid of the gastric juice can arrest this action, still he further

* De adipis concoctione et absorptione. Dorpati, 1860. Page 61.

observes "hic effectus succi gastrici, quo succi pancreatici actio in dissolvendos adipes turbatur, si acidum ejus sive natro, quod in bile inest, sive diversa via addito kali caustico neutrale redditur, r u r s u s t o l l i p o t e s t." As the chyme traverses the intestine, the gastric acid is gradually neutralized, and the pancreatic juice is then free to decompose the neutral fats. This gradual neutralization of the gastric acid has also its peculiar advantage, as it allows fresh and fresh portions of oxide of lipyl to be presented in *statu nascenti* to the biliary acids, in which state only combination appears to ensue. Thus it is provided that free biliary acid, pancreatic juice, and neutral fat should first be thoroughly intermingled, and only when enough bile has been secreted to completely neutralize the gastric acid does the influence of the pancreatic juice on the fats come into operation, followed by the immediate union of the oxide of lipyl with the biliary acids.

II. It is manifest that especial accessories are requisite to carry the fats through the watery blood fluid, as also to conduct them again out of the vascular system. It is further *à priori* probable that the bile presents the necessary requisites for these purposes, and that it not only itself passes in a soluble form into the blood, but imparts solubility in water to the fats and fatty acids. It is, indeed, true that a certain amount of fat is absorbed even when no bile reaches the intestine, but this is quite intelligible, as Beneke has found myelin, and at least one biliary constituent, in all parts of the animal organism, and even in the vegetable kingdom (*ut infra*).

III. The access of the bile only allows the absorption of a definite quantity of fat in each species of animal.

IV. Experiments on animals with biliary fistulæ show with certainty the importance of the bile, not only in the absorption of the fats, but for the general economy of the individual. All animals deprived of their bile could only be kept alive for a short period by the supply of enormous amounts of food, and all ultimately died of total inanition. If, now, a biliary constituent be essential to the production of myelin, and myelin be an essential component of nervous matter, this result is quite accounted for. The animals survive so long as the amount of myelin present in their food scantily suffices to supply that consumed in the organism. If these considerations be correct, myelin ought to be extracted largely by means of cold or hot alcohol from the contents of the intestine. This was actually the case. The meat supplied to the animals that were the subjects of experiment contained small quantities of myelin.

The extract prepared from the contents of the duodenum above the bile duct yielded but a trace of it, but the contents of the jejunum presented it in enormous quantities. Similar quantities were also obtained from the chyle.

It thus became of great moment to ascertain the relations of the biliary acids to glycerine. Both cholic and glycocholic acids were found to dissolve freely in glycerine, but the solutions had none of the characteristics of myelin. When treated with dilute mineral acids they became turbid, but only developed minute spherules. Almond oil, treated similarly, at first seemed to give an affirmative result, but subsequently was found unfit for experiment from the presence of preformed myelin; indeed, the difficulty of obtaining a fat quite free from this substance was for a time insuperable, till at last a pure stearin was prepared by repeated extraction with ether.

With this stearin the act of digestion was as nearly imitated as possible. To stearin kept at a temperature of 70° C. a small quantity of dilute hydrochloric acid was added, then quite fresh ox gall, then freshly expressed pancreatic juice, the whole being gradually neutralized by excess of potash solution. At the end of an hour, phosphoric acid was added in excess, and the mixture cooled. The stearic acid and a mass of whitish flocculi gradually rose to the surface. These were collected and treated for twelve hours with cold alcohol, then filtered and evaporated. A yellowish mass remained, which developed the myelin forms under the microscope. Ox gall was used in these experiments instead of the biliary acids, as experience had already pointed out cholesterine as a constant component of myelin. To at once obtain the right proportions of cholesterine and biliary acid it was thought advisable to resort to the ox gall. As initial experiments, the absence of myelin from ox gall was demonstrated, as well as its absence from a mixture of cholesterine and biliary acid. Thus the only conclusions that remain are either that the biliary acids unite with oxide of lipyl in *statu nascenti*, and that this combination, mixed mechanically with cholesterine, develops the forms of myelin, or that simultaneously with the union of the oxide of lipyl to the biliary acids, a combination of the liberated fatty acids with the cholesterine of the bile ensues, and so these forms are generated. The latter hypothesis Beneke regards as most unlikely. Even after ten hours' boiling of the cholesterine mother-liquor of the egg yolk with potash in alcoholic solution, no decomposition resulted. Thus the artificial production of myelin confirmed the view that it consisted of a combined biliary acid with oxide of lipyl mixed with cholesterine. It was, moreover, impossible to reproduce

the pure biliary acids from this combination just as they had failed to be obtained from the naturally produced myelin. It may well be asked whether this combination may not behave differently to other combinations of the oxide of lipyl, and may not be intended to enter into new associations in the economy, but never again to decompose into glycerine and biliary acid. In proportion to the amount of cholesterine added to the artificial myelin were the general characters of the shapes produced. The more cholesterine is added, the tougher and more resinous does the mass become, and simultaneously the microscopic forms increase in tenuity.

In a later series of investigations Prof. Beneke was enabled to obtain myelin from a number of products of the vegetable kingdom. Thus fine olive oil yielded it on boiling with potash, and extracting with alcohol. The young parts of a number of plants, such as the green leaves of fumaria, young asparagus, potato germs, the pollen grains of fumaria, and of lonicera tartarica alba also yielded it. It was also found pre-existing in great abundance in the ordinary pisum sativum. Simple extraction with cold alcohol for twenty-four hours produced it largely. The seeds of a great number of plants also yielded it, as of the white bean, rye, wheat, rape-seed, etc. Lastly, he obtained cholesterine from the pea and from olive oil.

ON MUSCULAR CONTRACTION.

By H. F. Baxter, M.R.C.S.

As the present inquiry must be considered a continuation of my previous investigations, I will briefly recapitulate some of the conclusions, partly deduced as corollaries from other conclusions, which I have been led to entertain in regard to muscular contraction. In a paper* "On the Polarized Condition of the Muscular and Nervous Tissues," it was stated, "During life, the muscular and nerve fibre may be considered as existing in a state of t e n s i o n, a f o r c e d state, and muscular relaxation may coincide, and would be synonymous, with this polarized condition or state of t e n s i o n and contraction, the result of a depolarization of the muscular fibre. In the normal state this d e p o l a r i z a t i o n is induced by means of

* Edinburgh New Philosophical Journal. April, 1858. Essay on Organic Polarity, chapter ix.

nervous agency; but it may also occur from other means, such as chemical or mechanical agencies, or whatever is likely to produce disorganization. Depolarization having occurred, the polarized state is easily and readily restored by the act of nutrition; hence contraction may be partly considered as the result of molecular attraction. The particles constituting the muscular fibre being in a state of self-repulsion, whilst in this polarized condition, would resist the force of molecular attraction unless depolarized; and, according to this view, the results of muscular contraction would necessarily follow, in conformity to the law as laid down by Schwann." In a subsequent investigation "on Muscular Power,"* I was forcibly struck with the importance of an elongated state of the muscle prior to contraction, the contractile power of a muscle increasing, up to a certain point, as the elongation of the muscle was increased.

The views that are at present entertained in regard to the influence of nervous agency over muscular contraction renders the question of muscular contractility an open one, and although my own opinion coincides with those who consider contractility as a property of the muscular fibre, independent of the nerve, still as the opposite opinion, that it is dependent upon nervous agency, is entertained by eminent physiologists, especially amongst the Germans, it behoves us to examine into the grounds upon which this conclusion is based. In discussing the question of muscular contraction it will, however, be necessary to bear in mind the two questions that are involved—1st, the contractility of the muscular fibre; and 2ndly, the contraction induced by nervous agency. The terms contractility and contraction, when employed to designate the same idea, appear to me to have created a great deal of confusion in the discussion of this subject; muscular contractility need not necessarily imply nervous agency, but muscular contraction may do so.

The greatest difficulty in solving these questions arises at the very commencement of our investigations, namely, the ignorance we are in as to the mode in which the muscular fibres are supplied with nerves. Microscopical anatomy has not yet decided this point for us; we only know that muscular fibres are freely supplied with nerves, and consequently the impossibility of deciding how far the means which we employ, whether chemical, mechanical, or electrical, for the purpose of exciting contractions, act first upon the nerves, or upon the muscular tissue alone, invalidates, to a great extent, any conclusion that

* Edinburgh New Philosophical Journal. October, 1863.

may be arrived at by their employment. For instance, if we use chemical or electrical means, the stimulating effect is not confined to the spot, but extends more or less over the surface of the muscle, and although Kuhne* has shown that a difference is observed in the effect produced by chemical agents, when applied to the trunk of the nerve or to the muscle, still the objection may be raised as to their limited action upon the muscular tissue. Upon these grounds I consider that the excitement of the muscle by mechanical means, such as the point of a needle or a piece of glass, would give more trustworthy results, but even here the same objections may be raised under the supposition that the surface of the muscle at the point of irritation may be connected with nervous tissue.†

If we could destroy the action of the nerve by paralysing it to its extreme ends without injuring the muscular tissue, a great point would be gained. It has been supposed, and a great many experimental results have been based upon this supposition, that this desirable object can be effected by means of passing an electric current, i n v e r s e l y, along the trunk of the nerve, producing the state which has been designated by Pflüger as a state of anelectrotone. In my present experiments I employed the following method:—

I took the two hind limbs of a middle-sized frog, separated from each other at the pelvis, and carefully dissected out the lumbar nerves down to the sciatic notch. The nerve of the limb *a* was then placed upon two strips of moistened bibulous paper, which rested on two distinct pieces of thick glass, the interval between them being about half an inch. Upon one end of each strip of paper rested the electrodes, which consisted of thick copper wire, and were so bent as to press firmly down upon its surface. The battery consisted of six of Grove's middling-sized cells; the sulphuric acid solution was formed with 1 part of strong acid to 12 of water, pure nitric acid being employed in the platinum cells. The limb *b* with its nerve was arranged exactly in the same manner as that of *a*, but reversed, so that the conditions of the two limbs should be the same. A current was now passed along the nerve *a* inversely, when a contraction occurred at the closing of the circuit in both limbs. The current was allowed to pass for twenty minutes, and during this period the nerves of both limbs were occasionally moistened slightly with distilled water, by means of a camel's hair pencil; an occasional contraction occurred in the limb *a*.

* Report on Muscular Contraction. By A. B. Duffin, M.D. In Beale's Archives of Medicine. Nos. X. and XI. January, April, 1862.

† The researches of Prof. Beale evidently show that the nerves cross the fibre, and form a plexus or net-work *external* to the sarcolemma.

After the passage of the current for twenty minutes, it was intermitted when tetanic contractions ensued in *a*, but upon renewing the current they subsided. I now carefully divided the skin covering the muscles of the thigh and the gastrocnemius in both limbs during the passage of the current, and then irritated the surface of the muscles by means of a pointed needle; the needle being inserted into a piece of glass tubing by means of a cork for a handle. A slight elevation was observed in the muscles of both limbs, at the point of irritation, and which gradually extended in a wave-like manner on each side, in the direction of the muscular fibre. The current was prolonged for thirty minutes, and the irritation repeated with the same results. The current was now suspended entirely, tetanic contractions ensued in the limb *a*, and which lasted, with gradual intermissions, for ten seconds. I now endeavoured to excite contractions by means of the needle, as before, but some time elapsed before any effect could be obtained in the limb *a*, and when it did occur, the swelling was persistent, there was not that wavy motion extending along the muscular fibre, as previously observed; with the limb *b* there was no difficulty in obtaining the contraction. Upon applying a drop of concentrated solution of common salt upon the surface of the muscles, slight fibrillar motions were produced in both limbs, but more extensive in *b*. When the salt was applied to the trunk of the nerves, contraction was produced in *b*, but not in *a*. The current from the battery was now sent along the nerve of limb *a*, direct, but no contraction; but when sent along the nerve of *b*, violent contractions. The current was then sent along the trunk of the nerve *a*, so as to traverse the limb, one electrode being in contact with the tendo Achillis. Slight contractions were now produced upon making and breaking the circuit.

Here, then, we had in these experiments destruction of the conducting power of the trunk of the nerve, still there was slight muscular contractions produced when the muscle was irritated with the point of the needle, and also when the chemical reagent was employed. Of course it will be said that the paralysing influence of the current, the so-called anelectrotonic state of the nerve, was not produced to its extreme ends, either during the passage of the current or the suspension of it; and that the slight effects which occurred when the current was suspended, and after the tetanic contractions had ceased, would show that this was the case. As a different view may be entertained in regard to this point, I will defer stating it until other experiments have been related.

The experiments were repeated on three other limbs in the same manner, but with this difference; in the first the current

was passed for half an hour; in the second for three-quarters of an hour; in the third for one hour. The muscles were irritated during the passage of the current and after the subsidence of the contractions as before. As my object was to obtain, if possible, a complete paralysis of the nerve, it will be unnecessary to enter into a detailed account of the whole experiment. In No. 1, during the passage of the current, there was no difference in the appearance of the muscular contraction when excited in the limbs. After the suspension of the current and when the tetanic contractions had subsided it was more difficult to excite the contraction in *a* than in *b*. In No. 2, during the current, the contraction excited in *a* presented a quivering motion; in *b*, the contraction was more prolonged and of greater extent. After the tetanic contractions had subsided the irritation in *a* produced a decided elevation, which continued for some time and then gradually subsided; in *b* a wavy motion of the fibre was first produced, and then a quivering motion. In No. 3 the irritation produced the same appearance in both limbs, viz., there was first an elevation and then an extended wavy motion on both sides. After the tetanic contractions had subsided in *a*, there was an elevation, and afterwards a slight quivering; whilst in *b* the elevation first arose, and then a wavy motion throughout the whole length of the fibre. The tetanic contractions lasted in No. 1 ten seconds, in No. 2 five seconds, in No. 3 eight seconds. No irritation of the trunk of the nerves, either by chemical or electrical means, produced contractions in *a*, unless the current was made to traverse the entire limb; but contractions were readily excited in *b* by these means.

From these results I can come to no other conclusion than that the passage of an inverse current along the trunk of the nerve is incapable of producing a paralysing effect to its extreme ends, but that a paralysis of its trunk may be induced, and which is dependent upon its disorganizing action, which takes place during its continuance; the disorganizing action, however, does not extend to the extreme ends. The difference in the contractions produced upon irritating the muscle after the subsidence of the tetanizing effects must, therefore, be due to other circumstances.

I now repeated the experiments on three other limbs, employing the current direct instead of inversely. In No. 1 the contractions excited in the two limbs were similar, producing an elevation and a wavy motion extending on both sides. Upon suspending the current there was a single contraction, but no tetanus; the contractions produced by irritating the muscle presented the same character in both limbs, and as

before. In No. 2 a slight quivering, which ultimately terminated in a prolonged wavy motion on each side of the irritated point occurred in *a*; whilst in *b* there was at first some difficulty in exciting contractions, but they then occurred and extended along the fibre. Upon suspending the current a slight quivering occurred in *a*, which soon subsided; upon irritating the muscle the quivering was renewed, then subsided, and a persistent elevation remained; in *b* contraction of the fibre was easily excited, and became more extended. In No. 3 the contractions excited in *a* and *b* were the same, an elevation occurred and then extended on both sides. Upon the suspension of the current there was no tetanus in *a*, but upon irritating the muscle a slight tetanus was produced, which lasted four seconds. Some difficulty occurred in arousing a contraction in *b*, it ultimately occurred, producing an elevation which became persistent.

I was now led, partly from these results and also from the researches of Matteucci in regard to the secondary electromotor power of the nerves, and from my own investigation respecting the so-called electrotonic state of the nerve,* to entertain the opinion that the effects referred to the so-called anelectrotonic state of the nerve, instead of being the result of a paralysis of the nerve extending throughout its whole extent, might be more immediately due to the state of the muscular fibre itself, the fibre, at the time, being in a state either of permanent contraction, or else that the contractile power of the muscular tissue itself had become completely exhausted from previous contractions. Matteucci has established the fact that the muscular current bears some relation to the contractile power of the tissue, and this has been confirmed by some of my own experiments. Here then we have a test which will enable us to judge how far the effects may or may not be referable to a loss in the contractile property of the tissue.

I now repeated the experiments, passing the current along the nerve *a* i n v e r s e l y for half an hour, three-quarters of an hour, and one hour, but in order to prevent the tetanic contractions from taking place upon the suspension of the current, the nerve trunk was divided low down the thigh before the circuit was broken, so as to avoid any contraction excepting that which might occur upon the division of the nerve. A section was then made through the thigh, leaving the lower half, which was separated from the knee joint. The skin was reflected, and thus a single electromoter muscular element was formed; a similar element was formed with the limb *b*, which had been traversed by the d i r e c t current.

* Edinburgh New Philosophical Journal. January, 1864.

In No. 1, when the section was made, there was slight quivering of the muscles in both limbs. They were then formed into a combined circuit with a galvanometer. The current indicated 3°. When each formed a single circuit, the amount was 1°; if anything it was in favour of limb *a*. A differential circuit was now formed; no result upon the needle.

In No. 2 similar quiverings were observed upon making the sections. The combined current was 4°; a single current, that of *a* 1½°, of *b* 2°. The differential current in favour of *b* 1°. This was a larger frog.

In No. 3 similar quiverings were observed, but not so prolonged. Combined current 3°; single current each 2°. Differential current slightly in favour of *b*.

In the following experiments the tetanic contractions were allowed to take place, and the circuits formed, after the contractions had ceased:—

In No. 1 the tetanic contractions lasted 20 seconds. On dividing the muscles they retracted in limb *a*; dividing the muscles of limb *b*, slight quiverings occurred. A circuit was formed with the limb *a*; no effect. With the limb *b* 3°; with the combined current 2°; with the differential current, 2° in favour of *b*.

In No. 2 the tetanic contractions lasted 18 seconds. On dividing the muscles, slight twitchings in those of *a*; in *b* merely retraction of the muscles. The current from *a* amounted to 1°; from *b* 3°. Combined current 3°; differential current 2° in favour of *b*.

In No. 3 the tetanic contractions lasted 10 seconds. On dividing the muscles of *a* an occasional quivering; dividing those of *b* the muscles merely retracted. The current from *a* scarcely affected the needle; from *b* 4°. Combined current 4°; differential current 3° in favour of *b*.

I now passed the current i n v e r s e l y along the nerve *a*, and, before suspending it, divided the nerve, to prevent the tetanic contraction from taking place. When the current was suspended, the muscles were irritated with the point of a needle. Contractions immediately took took place in the limb *a*, and to a greater extent than in those of *b*. When they had subsided, I could easily reproduce them in *a*. In another experiment, after the current had been passed for half an hour, I had some difficulty in exciting the contractions in *b*, whilst in *a* I could easily get them. The limb *b*, instead of being straight, the leg was bent upon the thigh, upon straightening the leg so as to stretch the muscles, and then irritating them, contractions ensued. I now passed the current along the nerve of another frog, and kept the leg flexed upon the thigh.

After dividing the nerve and suspending the current which had been passed for half an hour, slight quiverings of the muscles took place on irritating them. Upon stretching the limb, and then irritating the muscles, the contractions were much more powerful, and more readily obtained. I repeated this experiment, viz., kept the limb flexed during the passage of the current, so as to relax the muscles of the thigh and leg, the flexor muscles. Upon the cessation of the current, stronger and more extensive effects were obtained when the muscles were straightened and elongated by extending the leg than when they were flexed.

These latter experiments beautiful show the necessity of having the muscular fibre elongated, in order to obtain a contraction of the fibre; and the previous experiments indicate the connexion that exists between the electrical state of the tissue, and its contractile power. The electrical state of the muscle does not cause contraction, if anything, prevents it, by keeping the muscular fibre in a state of tension and elongation.

The current was now passed i n v e r s e l y along a nerve for half an hour. The limb was then suspended, and a weight (1 oz.) attached to the tendo Achillis to keep the muscles extended. Slight twitches of the muscles were observed on removing the weight for fifteen seconds; when the contractions had subsided, and then on irritating the muscle with the needle, no effect was observed. The experiment was repeated, and the nerve divided previous to suspending the current, to avoid the action of the nerve. The same weight was used, and after the lapse of fifteen seconds the muscle was irritated, slight quiverings were readily produced. The experiments were repeated, but the weights used were half an ounce and two ounces. The only difference in the results was this: when the nerve was not divided, a slight contraction was produced with the light weight, but none with the heavy weight. When the nerve was divided, contraction of the fibre was easily produced in the limb with the light weight, but with the heavier weight (two ounces) there was a persistent swelling, but no decided contraction.

These experiments show that when the trunk of the nerve which has been traversed by the current is allowed to remain, there is a constant source of excitement in the nerve to cause contraction, and which cannot be prevented by elongating the muscle so that the contraction should not take place. Let the cause of excitement be removed by dividing the trunk of the nerve, and separating it from the limb; the muscular fibre then retains its contractile power. If the weight, however, be too great for the muscle, so as to overstretch the fibre, the con-

tractile power is also destroyed. The question now occurs what effect has this stretching upon the muscular current?

I took the limbs of three frogs separated from the body, and to No. 1 attached 1 oz., to No. 2 1½ oz., to No. 3 2 oz., and suspended them for half an hour. On comparing the muscular current with those of their fellow-limbs no difference was observed, excepting in No. 3, which was somewhat less than that of the other. These results would appear to contradict the conclusion, that the electric state of the tissue bears a relation to the contractile power of the muscle. We should not, however, be justified in coming to this conclusion, for there is evidently a great difference between the loss of power arising from over-work, from constant contraction, and that arising from over-stretching of the muscular fibre. In the one case, during muscular action force is evolved from internal action; in the other there is no resistance to the extending force; in fact, there is no internal action. The results would be different if the limb was connected with the living animals when a resisting power would be called forth, so as to excite muscular action, as I have ascertained in a former paper.*

An intermittent current was now passed along a nerve for twenty minutes so as to keep the muscles in constant action, in a state of tetanus; on suspending the current and irritating the muscles, slight quiverings occurred. The nerve was divided and separated, the flexor muscles became contracted, and I could not excite contraction in them. I then stretched the limb, and allowed it to remain quiet for a short time—two minutes; upon irritating the muscle an elevation occurred which remained for some minutes; but when the limb was again stretched, I could not reproduce it. The experiment was repeated, but as the results were similar to those observed served when tetanus is produced after the passage of the i n v e r s e current, I need not detail them; there was the same loss in the muscular current as compared to that of the quiescent limb, and the same difficulty in obtaining contractions of the muscular tissue; the contractile power evidently became exhausted. And here I will just observe that the results recorded in the present paper have been obtained upon muscles in limbs separated from the animal, so that muscular action has been attended with a loss of power without the means of restoration, and I would have related some experiments showing the importance of the circulation of the blood in influencing the results, but having already done so in a former paper I must now refer to them.†

* On Muscular Power. Edinburgh New Philosophical Journal. October, 1863.

† Edinburgh New Philosophical Journal. October, 1863.

CASE OF PERFORATING ULCER OF THE DUO-DENUM. GALL STONES.

By Maxwell Mastiss, M.D.

The lady who was the subject of this note had been at various times under my care for symptoms indicative of ovarian disease, and at other times on account of obstinate constipation, vomiting and other evidences of disordered digestion. Active purgation, and enforced abstinence from spirits (to which liquor she was unfortunately far too partial) sufficed to relieve the vomiting. At no time were there any indications of the presence or passage of gall stones. On the 13th April, 1864, after having been for some months in pretty good health, she left home early in the morning, apparently quite well, but about noon, immediately after having taken some soup, was seized with violent pain in the abdomen, and returned to her residence. In the evening I found her writhing in great agony, the pain being referred especially to the umbilical region. Pressure and friction afforded some slight relief. The abdomen was scarcely tympanitic, and not at all tender on pressure. There had been slight vomiting, the bowels had not been relieved for two days, on which account she had taken a dose of castor-oil in the morning. The tongue was moist and covered with a thick white fur. Pulse 70, full and soft. Judging the case to be one of colic, I prescribed a cordial draught containing a little laudanum, ordered the application of a turpentine stupe to the belly, and the use of an enema. The latter brought away a few pieces of flattened fœces of the diameter of a goose quill. A second injection returned unchanged. The pain continued unabated till 4 A.M. the following morning, when a sudden change occurred. I saw her in ten minutes, but she was in a state of collapse, and she died in less than half an hour after the change was first noticed.

Post-Mortem.—Abdomen only examined. General peritonitis, and a large quantity of pus in the cavity of the belly. At the upper part of the duodenum, near the pylorus, was an ulcer of the size of a three-penny piece, the margins of which showed clear evidence of recent perforation, were stained with bile, and had small fragments of gall stone still adhering to them. Lying on the bowel, within an inch from the ulcer, was a black nodular gall stone of the size of a large pea, and much resembling a mulberry calculus in appearance. In the gall bladder itself were a great number of similar concretions, but none in the gall ducts. The intestines were empty and presented no indication of obstruction at any part. The liver and kidneys fatty. Both ovaries were diseased, the cysts were about the size of a large plum, and contained a dark grumous fluid. The peritoneum in their neighbourhood showed many old adhesions.

(*Continued from page* 108.)

Acute Rheumatism.—E. G., æt. 53. Cook. Adm. Mar. 9. Disch. Ap. 22. In H. 44 days. *Recovery.* Dr. Garrod, vol. 1, p. 63. Acute rheumatism 7 years ago. Prev. ill. 3 days. Ankles, knees, wrists, and shoulders affected in succession. Profuse sweating. Basic systolic bruit. Urine albuminous, containing granular casts. On 7th day, friction sound over heart (26 days) pain in. præcordia and dyspnœa. Pains about shoulders till 40th day.

Quinine and bicarbonate of potass (31 *days*). *Then syrup of iodide of iron. Leeches to præcordia.*

Acute Rheumatism.—F. F., æt. 28. Street Keeper. Adm. June 9. Disch. June 20. In H. 11 days. *Recovery.* Dr. Garrod, vol. 2, p. 159. Acute rheumatism 9 years ago. Chorea 1 month afterwards. Prev. ill. 1 month. On adm. pain in left shoulder, elbows, and knees. Free sweating. Tongue clean. Systolic bruit over apex of heart. Free from pain on June 18.

Bicarbonate of potass and quinine.

Rheumatism.—Dyspepsia.—M. W., æt. 44. Adm. Mar. 12. Disch. Ap. 1. In H. 19 days. *Recovery.* Dr. Beale, vol. 6, p. 119. Pains in joints, headache, emaciation. loss of appetite. On 11th day after adm. free from pain. Appetite good.

Quinine and bicarbonate of potass. Pepsine and hydrochloric acid.

Subacute Rheumatism.—W. F., 53. Porter. Adm. Mar. 21. Disch. Ap 7. In H. 17 days. *Recovery.* Dr. Beale. Five or six attacks during last 20 years. Prev. ill. 5 wks. Right wrist, left wrist, right knee and ankle, left knee and ankle. affected in succession. Sweating. Tongue slightly furred.

Alkaline mixture, and quinine and opium.

Subacute Rheumatism.—M. O., æt. 16. Servant. Adm. Mar. 17. Disch. May 27. In H. 71 days. *Recovery.* Dr. Garrod, vol. 2, p. 83. First attack. Prev. ill. 1 week. Pain and swelling of right knee. On fourth day pain in left shoulder. Sweating. P. 104. Knee well on 10th day. The shoulder remained painful for some time, and became stiff, so that she could not raise the arm. When discharged she had the full use of it.

Quinine and bicarbonate of potass (16 *days*). *Quinine, sulphate of iron, and potassium and ammonio citrate of iron. Passive motion of left arm.*

Rheumatism.—J. S., æt. 32. Butcher. Adm. Ap. 13. Disch. May 16. In H. 33 days. *Recovery.* Dr. Beale, vol. 6, p. 127. Acute rheumatism 3 months ago. Prev. ill. 6 wks. Commenced in ankles. Then in right knee, wrists, and shoulders. Loss of appetite. Sweating. On adm. ankles and wrists red and swollen, knees swollen and covered with a slight blush. Convalescent about May 5.

Bicarbonate of potass, opium and sulphate of magnesia, with inf. calumbæ. Alkaline lotion to joints. Afterwards blisters above knees, and bicarbonate of potass and liq. cinchonæ.

Rheumatism.—M. L., æt. 22. Servant. Adm. May 26. Disch. June 20. In H. 25 days. *Much relieved.* Dr. Garrod, vol. 1, p. 113. Previous illness 1 month. Pain and swelling of knees. On adm. right knee much swelled, left knee slightly swelled. Tongue clean.

Iodide of potassium. Iodine paint to knees.

Chronic Rheumatism.—J. P., æt. 17. Adm. Feb. 27. Disch. Ap. 22. In H. 54 days. *Relieved.* Dr. Johnson, vol. 16, p. 125. Typhus 10 years ago, followed by deafness. Acute rheumatism 8 months ago; heart not affected. During convalescence pain returned in left knee (3 wks.). Ten weeks ago left hip became painful, and has remained so ever since.

Iodide of potassium and citrate of iron. Blisters over hip. Subdermic injection of morphia. Hot air baths.

Chronic Rheumatism.—A. B., æt. 23. Servant. Adm. Feb. 21. Disch. Ap. 29. In. H. 67 days. *Relieved.* Dr. Johnson, vol. 16, p. 125. Housemaid's knee 2 years ago. Pain in joints and weakness ever since. On adm. wrists and small joints of fingers painful and swollen. Flatulence, loss of appetite, and sour eructations.

Iron and iodide of potassium. Afterwards iron and bicarbonate of potass. Blisters above wrists.

Rheumatoid Arthritis.—W. U., æt. 60. Clerk. Adm. June 1. Disch. June 27. In H. 26 days. *Relieved.* Dr. Garrod, vol. 1, p. 119. Temperate. No family history of gout. First attack, 13 yrs. ago in both great toes. An interval of 2 yrs. between first and second attack. Three or four slighter ones since. Prev. ill. 7 months. General gout commencing in knees. On adm. almost all the joints of extremities stiff and painful. No concretions. No uric acid in serum of blood drawn on the 20th day after adm.

Ammoniated tincture of guiacum and iodide of potassium.

*Gout.—G. G., æt. 48. Cabinet Maker. Adm. Mar. 23 Disch. Ap. 1. In H. 9 days. *Recovery.* Dr, Garrod, vol. 1, p. 78. Moderately temperate. First attack 9 years ago in ball of right great toe. The second 5½ years ago in both feet. 5 years ago a third in both feet. 3 years ago a fourth in hands and elbows. General gout twice in last six months. Prev. ill. 3 days. Right hand, right elbow, left knee, right knee, and feet affected in succession. On adm. right hand and elbow very red and swelled. No concretions. Liver large. Urine albuminous for 3 days after adm. Well on 5th day after adm.

Colchicum wine. Ammoniated tincture of guaiacum.

*Gout.—W. B., æt. 47. Cabman, Adm. Mar. 23. Disch. May 9. In H. 47 days. *Much relieved.* Dr. Beale. Not very temperate. Three attacks of gout in last 7 years, each commencing in right great toe. The last attack 2 yrs. ago. Prev. ill. 9 days. Commenced in ball of right great toe, then in right ankle, right knee, left ankle. No concretion in ears, ball of right great toe enlarged. Convalescent on 32nd day, with exception of slight redness and tenderness about left ankle.

Bicarbonate of potass. opium, sulphate of magnesia, and bark (36 *days*). *Sesquichloride of iron* (5 *days*). *Then bicarbonate of potass.*

Gout.—Disease of Bladder.—A. A., æt. 51, Brass Founder. Adm. Nov. 17. Disch. Nov. 30. In H. 13 days. *Relieved.*—Dr. Beale, vol. 7, p. 9. First attack of gout three years ago. Dysuria and irritable bladder, since acute renal dropsy 2½ years ago. Occasional hæmaturia 12 months. Lumbar pain 2 months. Urine albuminous, containing blood, phosphates, and bladder epithelium. Gout in left foot on 3rd day after adm.

Sesquichloride of iron. Bark, colchicum, bicarbonate of potass, and iodide of potassium. Loins dry cupped.

Gout.—G. P., æt. 44, Paperhanger. Adm. Dec. 17. Disch. Dec. 26. In H. 9 days. *Recovery.*—Dr. Garrod, vol. 3, p. 170. Formerly a House-painter. First attack 16 years ago. Since then lead colic twice, gout frequently. Now ill 2 days. Left thumb painful, anchylosis of ball of left great toe, and partially of left ankle. No concretions. Slight purpura. Well about 10th day.

Guaicum and iodide of potassium.

Gout.—J. P. M., æt. 38, Gunmaker. Adm. Oct. 31. Disch. Nov. 9. In H. 9 days. *Recovery.*—Dr. Garrod, vol. 3, p. 129. Gout three or four times a year for last seven years. Prev. ill. 14 days. In left knee, right foot, right knee, and right thumb. Moderate quantity of uric acid in blood serum. Slight albuminuria. Quite well on 22nd day.

Colchicum, and sulphate and carbonate of magnesia.

Gout.—J. D., æt. 59, Butcher. Adm. Sept. 22. Disch. Oct. 17. In H. 26 days. *Recovery.* Dr. Garrod, vol. 3, p. 35. Not very temperate. Gout frequently during last 20 years. Weakness of left arm, and wasting of muscles from disuse, consequent on gout in wrist, elbow, and shoulder during last 2 months.

Iron. Galvanism. J. P. to hand.

Gout.—W. D., æt. 38, Bootmaker. Adm. Sept. 7. Disch. Oct. 3. In H. 26 days. *Recovery.* Dr. Johnson, vol. 19, p. 46. Intemperate. Father gouty. First attack. Prev. ill. 7 days. In left hand, ankles, right hand, hips, and shoulders. Tongue slightly coated. Skin dry. Well on 26th day.

Bark, bicarbonate of potass, and colchicum. Cotton wool and alkaline lotion to joints.

Gout.—G. D., æt. 42, Coachman. Adm. Sept. 12. Disch. Sept. 28. In H. 16 days. *Recovery.* Dr. Johnson. Acute rheumatism 4 years ago. Pain in chest, shortness of breath, and loss of appetite, 5 days. Right great toe attacked on day before adm., then right foot, ankles, left hand and fingers, right wrist and fingers. Free sweating. Tongue furred. Free from pain on 11th day.

Bark, iodide of potassium, and bicarbonate of potass. Colchicum and opium.

Gout.—G. G, æt. 46, Bootmaker. Adm. Oct. 28. Disch. Dec. 19. In H. 52 days. *Recovery.* Dr. Garrod, vol. 3, p. 115. Mother had gout. Four months ago gout in 2nd and 3rd toes of left foot, right foot, right side, and right arm. Ill 9 weeks. Bladder irritable since. Prev. ill. 7 days. Pain in heels and ankles, back, right elbow and hand, left elbow. Tongue white. Trace of uric acid in blood serum. Free from pain on 35th day—hands afterwards painful.

Iodide of potassium and guaiacum. Quinine Colchicum. Cod-liver oil. E. L. to elbow. J. P. to hands.

Gout.—J. C., æt. 63, Salesman. Adm. July 11. Disch. Aug. 5. In H. 35 days. *Relieved.* Dr. Beale, vol. 5, p. 230. Father gouty. Frequent attacks for 25 years. Prev. ill. 5 weeks. Much effusion into left knee, left ankle, and right little finger also effected. Tongue furred. Concretion in right ear.

Bicarbonate of potass and sulphate of magnesia. Liq. cinchonæ. Blisters to knee.

Acute Rheumatism.—Kate L. æt. 34. Adm. Aug. 7. Disch. Sept. 9. In H. 33 days. *Recovery.* Dr. Garrod, vol. 1, p. 199. First attack 8 years ago. Prev. ill. 1 day. In right ankle, left ankle, knees, and hips. On 4th day right hand and wrist, and right elbow and shoulder attacked. On 6th day in left shoulder. Pain worse at night. Free sweating. Tongue coated. Violent noisy delirium on 6th evening. Free from pain on 8th day. The delirium began to subside about 19th day, and had quite vanished on 29th day. Heart normal throughout.

Quinine and bicarbonate of potass. Opiates and stimulants. Nitric acid and bark during convalescence.

*Acute Rheumatism.—A. R., æt. 14. Adm. Nov. 7. *Died* Nov. 30. Dr. Garrod, vol. 4, p. 2. First attack a year ago. Prev. ill. 3 days. Began with palpitation and cardiac pain. Then left knee and ankle right knee and ankle, and arm joints, attacked in turn. On adm. loud systolic bruit over apex, and constant severe præcordial pain. Tongue furred. Joints well on 11th day. On 18th day crep. and dulness at base of right lung, and slight albuminuria (6 days). On 19th day fresh pain in shoulders. On 21st day dulness and crep. at both bases. On 23rd day dulness, bronchial breathing, and bronchophony over both sides behind, nearly to scapular spine on right side. On 25th day P. 140, R. 80. On 27th day P. irregular, 136. Death from syncope. P. M.—Lower pulmonary

lobes nearly hepatized. Pericardium universally adherent. Beads of lymph on aortic valves. Left vent. hypertrophied.

Quinine and bicarbonate of potass. Bark and carbonate of ammonia. Brandy up to 12 *oz. Subdermic inj. of morphia reg. cord.*

Acute Rheumatism.—A. R., æt. 22, Compositor. Adm. Sept. 11. Disch. Oct. 17. In H. 36 days. *Recovery.* Dr. Garrod, vol. 3, p. 8. Four or five attacks since the first, 15 years ago. Prev. ill. 8 days. In back of neck, left arm and shoulder consecutively. Tongue slightly furred. Free from rheum on 16th day, with exception of slight pain in neck. On 22nd day slight epistaxis and hœmoptysis, and much blood in motions for a few days. Has phthisis.

Bicarbonate of potass and quinine (6 *days*). *Turpentine and gallic acid for hæmorrhage. Quinine and cod-liver oil.*

Acute Rheumatism.—Eliza N., æt. 17, Servant. Adm. Sept. 14. *Died.* Sept. 21. In H. 7 days. Dr. Johnson. First attack. Prev. ill. 2 days. In back of neck, left hip, shoulder, elbow, wrist, ankle, and knee, then in same joints of right side. Profuse sweating. Yellowish brown fur on tongue. Systolic bruit over heart. On 7th day rhonchus and sibilus over lungs, and crepitation at bases. Albuminuria. Death on 10th day. P.M.—Recent thickening of mitral valve. Bronchial mucous membrane inflamed. Congestion of lower lobes of lungs.

Acute Rheumatism.—W. G. æt. 31. Adm. July 28. *Died* on July 29. Dr. Johnson. Had been ill a week. On adm. delirious. Extremities cold. P. thready, 146. Profuse sweating. Pain in hips. To and fro sound over heart.

Acetate of potass, carbonate of ammonia, chloric ether, and quinine and opium. Brandy 24 *oz.*

*Acute Rheumatism.—H. B. æt. 72, Surgical Instrument Maker. Adm. Nov. 25. *Died* on Dec. 12. In H. 17 days. Dr. Johnson, vol. 10, p. 167. First attack. Prev ill about 14 days. First left ankle, then right ankle, red swelled, pitting and painful. Purpuric spots on legs. On 29th day ankles free from pain, but very œdematous, and albumen, blood, renal epithelium, and waxy and granular casts in urine. On 31st day extreme tenderness over right kidney. P. 36. Tongue brown. Sibilus over lungs. Death on 32nd day. P.M.—Fluid in thoracic cavities. Lungs œdematous. Aorta atheromatous and dilated. Right kidney large, congested, studded with hæmorrhagic spots and small cysts.

Bicarbonate of potass and iodide of potassium (12 *days*). *Quinine and sulphuric acid* (3 *days*). *Aromatic spirits of ammonia and decoction of bark* (2 *days*). *Sinapism to loins. Purgatives.*

*Acute Rheumatism.—W. B., æt 32, Timekeeper. Adm. Oct. 20. *Died.* on Dec. 11. In H. 52 days. Dr. Garrod, vol. 3, p. 98. Eighth attack. The first, 14 years ago. Prev. ill. 6 days. Left knee, left ankle, right knee, right ankle, right wrist and knuckles, left wrist and knuckles, shoulders, and tempors maxillary joints affected in turn. Tongue white. Systolic bruit over apex on adm. On 11th day joints well, crepitation below left nipple and angle of scapula, dulness and crepitn. at right base. Sputa rusty on 18th day. Albuminuria on 19th day. Diarrhœa. On 46th day dulness below right scapula, behind, coarse crepitation all over lungs; sputa still bloody. On 58th day *delirous*, p. 160. Death on 59th day. P.M.—Patch of pulmonary apoplexy in right lower lobe. Pericardium somewhat adherent. Mitral orifice contracted. Beads of lymph on thickened edge of valve. Albuminous (?) degeneration of lower half of left kidney.

Bicarbonate of potash and quinine (2 *days*). *Acetate and carbonate of ammonia* (17 *days*). *Carbonate of ammonia* (18 *days*). *Bark and ammonia. Brandy to* 18 *oz. Astringents and opium for diarrhœa.*

*Acute Rheumatism.—W. B., æt. 27, Carpenter. Adm. June 3. Disch. July 22. In H. 49 days. *Recovery.* Dr. Beale, vol. 5, p. 178. First attack a

year ago. Prev. ill. 5 days. Left wrist, left shoulder, ankles and knees affected consecutively. Joints free from pain on 26th day. Return in shoulders. Finally well on 30th day. Heart normal on adm. Pericardial friction sound on 9th day (8 days). Cardiac dulness increased till 13th day, normal on 17th day. Systolic bruit after 22nd day. On 9th day dulness and small crepitn. at *right* base, afterwards œgophony, pleuritic rub, and absence of vocal vibration. On 15th day bronchial breathing (13 days). On 12th day dulness and crepitn. below *left* scapular angle. Br. Br. (17 days) on 15th day extending up to spine on 19th day. Lungs clear on 41st day. Expectoration untinged throughout. Albuminuria on 9th day (5 days).

Bi arbonate of potass (5 days). Liq. ammon. Acetatis (11 days). Sp. ammon. arom. (10 days). Muriatic acid and pepaine. Chloric ether and brandy. Cupped to 4 oz. over rt. side on 11th day. Dry cupping. Turpentine stupes.

Acute Rheumatism.—F. P., æt. 19, Adm. Nov. 3. In medical ward 29 days. *Reli ved.* Dr. Johnson, vol. 19, p. 135. Gonorrhœa 3 weeks. Prev. ill. 4 days. On adm. much effusion into left knee, right elbow painful. Tongue furred. Free sweating. Basic pericardial brush. Elbow well on 11th day. About 22nd day knee much larger, erysipelatous patch on inner side, much constitutional disturbance. Transferred to surgeons about Dec. 2.

Bicarbonate and nitrate of potass, and quinine, and opium (8 days). Then same with potass. Iodid. Leeches to knee. Purgatives. Straight splint.

Acute Rheumatism.—E. V., æt. 51, Charwoman. Adm. Sept. 26. Disch. Nov. 14. In H. 49 days. *Recovery.* Dr. Johnson, vol. 19, p. 91. First attack. Father gouty. Prev. ill. 11 days. In right hand knuckles and wrist, left knuckles and wrist, shoulders, right knee and ankle, left ankle. Free sweating. Tongue furred. Dyspnœa for 4 days before adm.; then cardiac pain and tenderness, and to and fro sound at apex. On 19th day only occasional friction sound at base; systolic bruit loudest at apex. Convalescent about 54th day.

Bicarbonate and nitrate of potass, and quinine, and opium. Brandy up to 8 oz. Iron.

Acute Rheumatism.—H. S., æt. 32, Compositor. Adm. Oct. 14. Disch. Nov. 14. In H. 31 days. *Recovery.* Dr. Johnson, vol. 17, p. 126. First attack. Prev. ill. 14 days. In left knee, right knee, ankles, and hips, right shoulder, wrist, and hand successively. Profuse sweating. Tongue moderately furred. First sound of heart prolonged. Free from pain on 42nd day.

Bicarbonate and nitrate of potass, and quinine opium. Iron. Brandy.

Acute Rheumatism.—A. P. 16, Compositor. Adm. July 20. Disch. Sept. 4. In H. 46 days. *Recovery.* Dr. Beale, vol. 6, p. 234. Acute renal epilepsy 9½ months ago. First attack. Prev. ill. 14 days. In left instep, left knee, right ankle, right elbow, left elbow, right knee, hands, wrists, and right sterno clavicular joint. Free sweating. Creamy fur on tongue. Slight albuminuria on adm. Cystine in urine about 37th day. Slight pain in joints on 40th day. Heart unaffected.

Bicarbonate of potass, and liq. ammon. acetatis.

Acute Rheumatism.—W. W., æt. 34, Stone Sawyer. Adm. Oct. 19. Disch. Nov. 11. In H. 23 days. *Recovery.* Dr. Johnson, vol. 17, p. 134. First attack. Prev. ill. 5 weeks. All joints affected. Free sweating. Soft systolic bruit at apex, and faint pericardial brush at base. Free from pain on day before adm. Had been under treatment a fortnight.

Bicarbonate and nitrate of potass, carbonate of ammonia, and quinine and opium.

Acute Rheumatism.—A. H., æt. 28, Glazier. Adm. Aug. 5. Died on Aug. 7. In H. 2 days. Dr. Beale, vol. 5, p. 194. First attack. Prev. ill. 14 days. Began in knees. Most of the joints attacked. On adm. friction sound at base of heart. Delirium. Diarrhœa. Skin hot and dry. Died on 7th.

Bicarbonate and nitrate of potass, with liq. opii. sed., and quinine and opium.

Acute Rheumatism.—M. M'C., æt. 48, Shoemaker. Adm. June 1. Disch. July 15. In H. 44 days. *Recovery.* Dr. Johnson, vol. 16, p. 229. First attack. Prev. ill. 12 days. In right elbow, right hand, left hand and shoulder, toes and and ankles successively. Tongue thickly coated. Profuse sweating. Pericardial friction sound on 15th day. Free from pain on 22nd day. Slight return in shoulders and feet. Finally free from pain on 45th day.

Bicarbonate of potass and carbonate of ammonia, with quinine and opium. Brandy to 8 oz. Opiates. During relapse acetate of potass instead of bicarbonate.

Acute Rheumatism.—R. T., æt. 39, Keeper at an asylum. Adm. June 8. Disch. July 22. In H. 44 days. *Recovery.* Dr. Johnson, vol 17, p. 47. Fourth attack. The first 16 years ago. Gout 13 months ago. Prev. ill. 5 weeks. Ankles, knees, shoulders, elbows, and wrists attacked in succession. Profuse sweating. Brown fur on tongue. Friction sound over heart's base from 38th to 52nd day. Much crepitus at bases of lungs about 47th day. Joints well on 45th day. Relapse (10 days) about 66th day.

Bicarbonate of potass and quinine and opium (16 *days*). *Brandy to* 8 *oz.*

Acute Rheumatism.—A. C., æt. 26, Porter. Adm. Nov. 24. Disch. Dec. 15. In H. 21 days. *Recovery.* Dr. Garrod, vol 3, p. 160. Gout 8 years ago. Ankles swelled 1 mo., painful 14 days. In left ankle, right ankle, knee and hip successively. Free sweating. Tongue coated. Systolic bruit at apex on adm. Crepitus at bases of lungs. Free from pain on 24th day.

Quinine and bicarbonate of potass. Brandy. Turpentine stupes to chest.

Acute Rheumatism.—P. R., æt. 25, Cook. Adm. Nov. 6. Disch. Nov. 21. In H. 15 days. *Recovery.* Dr, Johnson, vol. 17, p. 145. First attack. Prev. ill. 2 days. In right wrist, then in ankles. Free sweating. Sore throat and dysphagia. Free from pain on 8th day.

Chlorate of potass, sesquichloride of iron, and glycerine. Iron and glycerine to throat.

Acute Rheumatism.—R. L., æt. 38, Waiter. Adm. Oct. 21. Disch. Nov. 4. In H. 14 days. *Recovery.* Dr. Johnson, vol. 18, p. 61. First attack 8 yrs. ago. Prev. ill. 14 days. Ankles and right knee affected. Tongue white. Free from pain on 24th day. Headless tapeworm expelled after a dose of malefern oil.

Bicarbonate of potass, with iodide of potassium and bark. E. L. to knee.

Acute Rheumatism.—Alice S., 10. Adm. Oct. 31. Disch. Nov. 18. In H. 18 days. *Recovery.* Dr. Johnson, vol. 17, p. 77. Second attack. Prev. ill. 5 days. Ankles, knees and left wrist affected successively. Basic systolic bruit. P. 92. Free from pain on 18th day of illness.

Bicarbonate and nitrate of potass, and quinine and opium.

Acute Rheumatism.—W. P., 18, Shopman. Adm. Oct. 20. Disch. Oct. 31. In H. 11 days. *Recovery.* Dr. Johnson, vol. 18, p. 51. First attack a year ago, slight. Prev. ill. 3 wks. All joints attacked before adm. Then knees painful. Tongue furred. Mitral systolic bruit at apex. Free from pain about 22nd day.

Bicarbonate of potass, quinine, and opium.

Acute Rheumatism.—Eliza B. 21, Servant. Adm. Oct. 28. Disch. Nov. 18. In H. 21 days. *Recovery.* Dr. Beale, vol. 5, p. 334. First attack. Prev. ill. 14 days. In back, knees, ankles, right wrist, left wrist, elbows, shoulders, successively. Free sweating. Tongue slightly furred. Soft systolic bruit at apex, and pericardial friction sound on adm. Free from pain on 23rd day.

Bicarbonate of potass and sp. ammon. arom. (13 *days*). *Brandy* 4 *oz.*

Acute Rheumatism.—Mary R., æt. 30, Cook. Adm. June 10. Disch. July 18. In H. 38 days. *Recovery.* Dr. Garrod, vol. 1, p. 136. First attack 9 years ago,

heart affected. Second 3 months back, more or less since. Prev. ill. 5 days. Hands, elbows, shoulders, ankles, knees affected successively. Tongue coated. Much sweating. Old mitral systolic bruit. On 9th day sputa slightly bloody, and dulness crepitus, and a little bronchial br. at bases. Then free from pain, but slight return afterwards in left wrist.

Quinine and bicarbonate of potass. Opiates nightly. Hirud. viij. reg. cord.

Acute Rheumatism.—Fanny H., æt. 22, Servant. Adm. Oct. 14. Disch. Nov. 11. In H. 28 days. *Recovery.* Dr. Garrod, vol. 3, p. 77. First attack 10 years ago. Prev. ill. 6 days. In right knee, right hand, elbow and shoulder, and right ankle, then in joints of left side in same order successively. Free sweating. Tongue slightly furred. Systolic bruit over apex on adm. Friction sound over base on 8th day. Free from pain on 28th day.

Bicarbonate of potass and quinine (22 days).

Acute Rheumatism.—J. D., æt. 17, Housemaid. Adm. Oct. 20. Disch. Nov. 14. In H. 25 days *Recovery.* Dr. Garrod, vol. 3, p. 104. First attack. Prev. ill. 7 days. In ankles, knees, elbows, and wrists successively. Basic pericardial friction sound on adm. Tongue furred. Skin hot. Much pain in chest (till 22nd day). Joints well on 15th day.

Quinine and bicarbonate and acetate of potass (16 days). Brandy.

Acute Rheumatism.—E. C., æt. 29, Ostler. Adm. June 13. Disch. July 8. In H. 25 days. *Recovery.* Dr. Johnson, vol. 15, p. 247. First attack 8 yrs. ago. Lead colic 4 yrs. ago. Prev. ill. 4 days. In right ankle, left ankle, knees, left shoulder, and wrists successively. Profuse sweating. Yellowish white fur on tongue. Bowels confd. Systolic bruit over apex of heart on adm. Free from pain on 20th day.

Acetate of potass and carbonate of ammonia, and quinine and opium. Brandy.

Acute Rheumatism.—G. D., æt. 20, Porter. Adm. Sept. 21. Disch. Oct. 31. In H. 40 days. *Recovery.* Dr. Garrod, vol. 3, p. 31. First attack. Prev. ill. 10 days. In ankles, shoulders, elbows, wrists, fingers, and sterno clavicular joints successively. Tongue furred. Moderate sweating. Systolic bruit over apex on adm. Free from pain on 20th day, but fresh attack on 25th day in ankles, knees, and wrists.

Quinine and bicarbonate of potass.

Acute Rheumatism.—Anu R., æt. 26, Married. Adm. Sept. 30. Disch. Oct. 19. In H. 19 days. *Recovery.* Dr. Garrod, vol 3, p. 46. First attack a year and a half ago. Prev. ill. about 25 days. Left knee and ankle, right knee and ankle, left shoulder, elbow and wrist, and right shoulder, elbow and wrist affected successively. Systolic bruit over heart on adm.; loudest at apex. Tongue furred. Frequent metastasis. Friction sound at base about 28th day. Free from pain on 32nd day.

Quinine and bicarbonate of potass. Brandy.

Acute Rheumatism.—F. A., æt. 16, Housemaid. Adm. Aug. 6. Disch. Sept. 23. In H. 48 days. *Recovery.* Dr. Beale, vol. 5, p. 240. Third attack. First 5½ yrs. ago. Prev. ill. 12 days. In ankles, knees, shoulders, elbows, and wrists successively. Constant præcordial pain. Free sweating. Tongue thickly furred. P. 120. Systolic bruit over apex on adm. Free from pain on 48th day.

Quinine, bicarbonate, and nitrate of potass, and acetate of ammonia.

*Acute Rheumatism.—Alice J., æt. 16, Servant. Adm. June 27. *Died* on Aug. 2. In H. 36 days. Dr. Beale, vol. 5, p. 210. First attack four years ago. Prev. ill. 3 days. Knees, ankles, and right hand painful in turn. Profuse sweating. Thick white fur on tongue. Acute præcordial pain. Slight albuminuria. On 7th day diarrhœa and tympanites (3 days), and crepitation and pleuritic rub at right base. Afterwards dullness and bronchial br. over bases till 32nd day, when dulness, no br. br. or resp. murmur, slight crepitation. Joints well on 12th day.

About 18th day hœmaturia and œdenia of feet and ankles. To and fro sound over heart on adm. About 13th day extension of cardiac dulness to 2nd rib and right sternal edge; disappearance of friction sound. Rub again heard on 14th day, all over heart on 16th day. Great increase of cardiac dulness afterwards. P. M. Recent pleuritic adhesions. Three pints fluid in pericardium. Fresh adhesions about base of heart. Fibrinous warts on mitral valve. Right lung and lower lobe of left dark red and solid.

Bicarbonate of potass, acetate of ammonia and sp. ammon. arom. Afterwards sulphuric ether, sesquichloride of iron, and liquor cinchonæ. Brandy to 16 oz. Mercurial ointment with opium over heart. Loins dry cupped.

Acute Rheumatism.—Sarah C., æt. 24, Married. Adm. July 10. Disch. July 24. In H. 14 days. *Recovery.* Dr. Garrod, vol. 1, p. 167. First attack six years ago. Prev. ill. 4 days. Knees, ankles, and hips affected. Moderate sweating. Tongue blankety. P. 104. Free from pain on 11th day.

Quinine and bicarbonate of potass.

Acute Rheumatism.—Eliza K., æt. 25, Servant. Adm. June 1. Disch. July 1. In H. 30 days. *Recovery.* Dr. Johnson, vol. 16, p. 233. First attack 13 years ago. Ankles painful 2 months. Prev. ill. 1 week. Ankles, knees, wrists, shoulders, and elbows successively affected. Profuse sweating. Furred tongue. Slight basic systolic bruit. Free from pain on 19th day.

Bicarbonate of potass and carbonate of ammonia, and quinine and opium.

Acute Rheumatism.—Jane T., æt. 22, Servant. Adm. June 17. Disch. July 1. In H. 14 days. *Recovery.* Dr. Garrod, vol. 2, p. 178. First attack. Prev. ill. 5 days. Ankles, knees, wrists, and elbows attacked. Tongue furred. Systolic bruit over apex on adm. Free from pain on 10th day.

Quinine and bicarbonate of potass.

Acute Rheumatism.—L. K., æt, 16, Kitchenmaid. Adm. Sept. 15. Disch. Oct. 5. In H. 20 days. *Recovery.* Dr. Garrod, vol. 3, p. 16. First attack. Prev. ill. 8 days. Left ankle, knee, elbow, and hand, right shoulder, elbow, and hand successively affected. Free sweating. Tongue furred. First sound of heart prolonged, and slight præcordial pain. Free from pain except in left hand on 23rd day.

Quinine and bicarbonate of potass. Opiates at night. Brandy.

Acute Rheumatism.—D. O., æt. 19, Waiter. Adm. Oct. 30. Disch. Nov. 21. In H. 22 days. *Recovery* Dr. Garrod, vol. 3, p. 123. First attack 4 years ago. Prev. ill. 4 days. In right ankle and left wrist. Slight sweating. Tongue furred. Free from pain on 9th day. Roughness with second sound about 11th day. P. intermittent on discharge.

Quinine and bicarbonate of potass.

Acute Rheumatism.—T. C., æt. 38, Cabman. Adm. May 18. Disch. July 13. In H. 57 days. *Recovery.* Dr. Johnson, vol 13, p. 39. First attack 11 years ago. Prev. ill. 1 day. Left knee and ankle, right hand and wrist, and right knee and ankle affected in succession. Profuse sweats. Tongue slightly furred. On 4th day friction sound over heart (20 days), and afterwards a faint systolic bruit. Free from pain on 33rd day. Relapsed on 44th day. Finally convalescent on 53rd day

Bicarbonate of potass and quinine and opium. Cotton wool to joints.

Acute Rheumatism.—Hope C., æt. 28, Married. Adm. Oct. 19. Disch. Nov. 14. In H. 26 days. *Recovery.* Dr. Beale, vol. 5, p. 320. Fifth attack; first 24 years ago. Prev. ill. 3 months. In left great toe, knees, hands, and elbows successively. Return of pain shortly after adm. Tongue furred. Free sweating. Double bruit at base; heart's impulse heaving.

Liquor cinchonæ and bicarbonate of potass. Whisky 6 oz.

Acute Rheumatism.—E W., æt. 38, Needlewoman. Adm. Oct. 5. Disch. Nov. 21. In H. 47 days. *Recovery.* Dr. Garrod, vol. 3, p. 59. First attack. Prev. ill. 14 days. On adm. very weak and emaciated, bedsore over sacrum.

P. 124, R. 36. Cardiac dulness increased in area; to and fro sound at apex; slight albuminuria. On 3rd day after adm. right shoulder and wrist, left ankle, elbows and knees painful. Joints soon well, but heart's action irregular till 27th day.

Bicarbonate of potass, carbonate of ammonia, and bark. Brandy.

Acute Rheumatism.—A. L., æt. 33, Laundress. Adm. Nov. 17. Disch. Dec. 30. In H. 43 days. *Recovery.* Dr. Johnson, vol. 18, p. 98. First attack. Rheumatic iritis 2 years ago. Prev. ill. 5 weeks. Ankles, wrists, knuckles, and right knee successively affected. Selerotitis about 48th day. On 64th day free from pain except in right ankle. Finally free from pain 2 days before discharge.

Bicarbonate of potass, iodide of potassium, and bark. Opium. E. L. post. aur. dext.

Acute Rheumatism.—S. Ann de K., æt. 15. Adm. Aug. 12. Disch. Oct. 16. In H. 65 days. *Recovery.* Dr. Johnson, vol. 17, p. 115. Prev. ill. 4 days. Left foot and knee, right ankle and knee, shoulders, elbows, and wrists successively affected. Thick blankety fur on tongue. Free sweating. Loud basic diastolic bruit. Pericardial friction sound on 7th day, quite disappearing as cardiac dulness increased, again audible on 34th day. About 13th day delirium at night, and dulness and pleuritic rub at left base. Remained very anæmic.

Quinine and bicarbonate of potass (13 *days*). *Then liq. ammon. acetatis and sp. ammon. arom. Iron. Brandy up to* 12 *oz.*

Acute Rheumatism.—Louisa C., æt. 25, Married. Adm. Nov. 14. Disch. Dec. 9. In H. 25 days. *Recovery.* Dr. Beale, vol. 7, p. 1. Had scarlet fever 13 years ago. Three weeks ago sore throat, enlarged cervical glands, and fever without eruption. Right thumb and arm, back of neck, left arm, knees and ankles affected successively during last 14 days. Free sweating on adm. General cuticular desquamation. Elbows, right shoulder, and knees painful. Tongue slightly furred. Basic systolic bruit. Free from pain on 25th day.

Bicarbonate and nitrate of potass. Occasional nightly opiates.

*Acute Rheumatism—Pyæmia—Erysipelas.—P. K., æt. 21, Policeman. Adm. April 14. *Died* on July 25. In H. 102 days. Dr. Beale, vol. 6, p. 28. Prev. ill. 1 month. Back of neck, legs, and shoulders painful in turn. Free sweating. Tongue furred. About beginning of May abscesses formed in calves of legs, followed by extensive sinuses. Much effusion into left knee about beginning of July, and about July 14 abscesses about right wrist and hand and head of right fibula. On July 17 erysipelas of face, followed by sloughing of skin of forehead and eyelids. Occasional delirium. Dry brown tongue. Fresh abscesses about knees and elbows and palm of left hand. Death.

Alkalies and quinine. Sesquichloride of iron and quinine. Cod liver oil. Pepsine. On July 4. *Potass, bicarb., eth. chlor., and sp. ammon. arom.* (10 *days*). *Then sp. amm. arom. and chloric ether. Brandy. Abscesses opened.*

Chronic Rheumatism.—A. R., æt. 19½, Warehouseman. Adm. Sept. 22. Disch. Nov. 11. In H. 50 days. *Much relieved.* Dr. Garrod, vol. 3, p. 37. Sequel of acute attack 9 months ago. Now fingers and ankles painful, impaired motion of elbows and right shoulder. On disch. ankles better.

Bark and iodide of potassium. Quinine and iron. Guaiacum. Gallic acid. Cod liver oil. Blisters to ankles.

Chronic Rheumatism.—W. J., æt. 42, Carpenter. Adm. Dec. 9. Disch. Dec. 19. In H. 10 days. *Relieved.* Dr. Beale, vol. 7, p. 27. Five attacks of acute rheumatism, the first 26 years ago. Prev. ill. 2 months. All joints affected. Increased cardiac impulse, and slight diastolic bruit.

Liquor cinchonæ and bicarbonate of potass.

Chronic Rheumatism.—W. R., 49, Coachman. Adm. Nov. 25. Disch. Dec. 16. In H. 21 days. *Very much relieved.* Dr. Johnson, vol. 17, p. 167. Frequent

acute rheumatism since first attack 44 years ago. Flying pains in joints since recovery from last attack 7 weeks ago. Prev. ill. 2 days. Left index, knuckle, and right hip painful and swollen. Slight sweating.

Bicarbonate of potass and iodide of potassium. Iron and colchicum afterwards added.

Chronic Rheumatism.—G. P., æt. 33. Warehouseman. Adm. Nov. 18. Disch. Dec. 28. In H. 40 days. *Improved.* Dr. Beale, vol. 6, p. 343. Acute rheumatism 4 years ago. Afterwards pain in hips. In various hospitals since. Now thighs painful and cannot be quite straightened on trunk. Has albuminuria.

Bark and iodide of potassium. Alkaline baths.

Subacute Rheumatism.—H. P., æt. 26, Cook. Adm. July 17. Disch. Aug. 26. In H. 40 days. *Recovery.* Dr. Garrod, vol. 1, p. 181. Ill three weeks. In elbows and left instep. On adm. pain and swelling of right elbow, extending up and down arm. Moderate sweating. Tongue furred. Basic systolic bruit. Free from pain on 8th day after adm. Elbow stiff for some time.

Guaiacum and iodide of potassium. E L. to elbow. Passive motion.

Subacute Rheumatism.—Mary A. R., æt. 24, married. Adm. July 9. Disch. July 22. In H. 13 days. *Recovery.* Dr. Garrod, vol. 1, p. 169. Third attack of rheumatism. First 8 years ago. Left ankle painful 5 months, stiff and swollen 2 months. Right hand painful 1 week. Left ankle and lower half of leg ædematous. On disch. scarcely any pain, ankle useful.

Quinine and bicarbonate of potass.

Subacute Rheumatism.—E. O., æt. 21, Housemaid. Adm. Sept. 24. Disch. Oct. 24. In H. 30 days. *Recovery.* Dr. Beale, vol. 5, p. 294. First attack. Prev. ill. 1 week. Right knee, left knee, left wrist and hand, right elbow, right ankle, and right knee (2nd time) attacked in turn. Sweating scanty. Tongue slightly furred, free from pain after 20th day.

Bicarbonate and nitrate of potass, and quinine and opium.

Rheumatoid Arthritis—H. A., æt. 53, Widow. Adm. Oct. 13. Disch. Oct. 24. In H. 11 days. *Relieved.* Dr. Garrod, vol. 3, p. 73. Ill 6 years. In right knee, hand, and shoulder. About 1½ years ago joints of left side affected in same order.

Guiacum, bark, and iodide of potassium.

Rheumatoid Arthritis.—Esther L., æt. 23, married. Adm. Oct. 29. Disch. Nov. 11. In H. 14 days. *Much relieved.* Dr. Garrod, vol. 3, p. 119. Prev. ill. 6 weeks. Wrists, finger-joints, right shoulder, elbows, and ankles stiff, painful, and worse at night. Has phthisis; right apex affected. Ankles improved.

Liq. cinchonæ, and iodide of potassium; then guaiacum and bark. Cod liver oil.

Rheumatoid Arthritis.—C. D. æt. 36, Monthly Nurse. Adm. June 26. Disch. Aug. 1. In H. 36 days. *Very much relieved.* Dr. Beale, vol. 6, p. 212. Prev. ill. 9 months. Laid up most of that time. Worse last fortnight. First in right great toe and ankles. On adm. right shoulder, elbow, hand, and wrist; left elbow, hand, knee, and ankle affected. Sweating freely. Creamy fur on tongue. Nearly free from pain on 19th day after adm.

Sp. ammon. arom. and Liq. ammon. acetatis. Opiates at night. Aperients.

Inflamed Knee.—Rhoda V., æt. 37, Servant. Adm. July 31. Disch. Aug. 12. In H. 12 days. *Recovery.* Dr. Garrod. Inflammation of right knee joint 3 weeks. Free from pain on Aug. 11.

Iodine paint. Passive motion. Infusion of quassia.

Diseases of Knee.—M. M., æt. 17, Kitchenmaid. Adm. Dec. 28. Disch.

Dec. 31. In H. 3 days. Dr. Johnson. Pain and great swelling of right knee, 1 week. Scrofulous Keratitis 3 weeks. Transferred to surgeons Dec. 31.

Gonorrhœal Rheumatism.—J. J. æt. 22, Clerk. Adm. Nov. 9. Disch. Dec. 12. In H. 33 days. *Recovery.* Dr. Garrod, vol. 1, p. 228. Gonorrhœa well 8 or 9 days ago. Prev. ill. 10 days. In left knee, hip, and sole of foot. Tongue slightly furred. First sound of heart prolonged. Hip and heel still slightly painful on 40th day.

Guaiacum and iodide of potassium. Asafœtida. Subdermic injection of morphia. Hot air baths.

Sciatica.—R. B., 40, Brickmaker. Adm. Sept. 1. Disch. Sept. 23. In H. 22 days. *Recovery.* Dr. Johnson. Second attack. First slighter, 12 months ago. Prev. ill. 10 weeks. Sudden severe pain from left loin to ankle. Loin tender. Leg numb. Free from pain on 20th day after adm.

Lumbago.—A. P., æt, 27, Footman. Adm. June 20. Disch. July 11. In H. 21 days. *Nearly well.* Dr. Johnson. Pain in loins very severe at night, and constipation 2 months. Oxaluria.

Aperients. Quinine and iron. Iodide of potassium and iron. Opiates. Hot air baths.

Pericarditis.—J. H., æt. 54, Labourer. Adm. July 21. Disch. Aug. 19. In H. 29 days. *Recovery.* Dr. Johnson, vol. 19, p. 26. Never had rheumatism. Pain in left shoulder 14 days. Pain in left side, dyspnœa, and nocturnal palpitation, 2 days. On adm. cardiac dulness much enlarged, and loud to and fro sound over heart. P. 100, R. 32. Dulness and crepitation under left clavicle, and at left posterior base. 10 days later heart's sounds and dulness normal.

Carbonate of ammonia and liquor ammon. acetatis (7 days). Then quinine and sesquichloride of iron. Brandy, 4 oz.

*Pericarditis.—T. K., æt. 46, Porter. Adm. Dec. 10. Disch. Dec. 30. In H. 20 days. *Recovery.* Dr. Johnson, vol. 19, p. 182. Intemperate. Typhus 10 years ago. "Caught cold" 2 weeks ago. Prev. ill. 3 days. On adm. pain and tenderness in left hypochondrium, cardiac dulness from 2nd rib to lower border of 6th, and to right of sternum. Friction (to and fro) sound near left nipple, very distinct, 7 days later, inaudible 11 days later. Dulness over lower part of left lung, fine crep. below the 5th rib, in front. Pleuritic rub, harsh br., and increased vocal thrill at angle of scapula. P. 120, R. 30. Expectoration scanty and untinged.

Carbonate of ammonia, chloric ether, quinine and opium (15 days.) Then quinine and sesquichloride of iron. Brandy, 6 oz.

Pericarditis.—H. F., æt. 39, Picture Frame Maker. Adm. Oct. 19. *Died* on Nov. 2. In H. 14 days. Dr. Garrod, vol. 3, p. 93. Cough and dyspnœa, at times, 12 years. Hæmoptysis 16 days ago. On adm. pallid. Systolic bruit over apex of heart; P. 96, R. 32; delirium at night. 3 days later loud to and fro sound over whole cardiac region (till death); 4 days later dulness and br. br. below angle of left scapula; 5 days later tongue dry, much delirium. P.M.—Bloody serum in pericardium. Much recent lymph on heart. Mitral orifice contracted. Left ventricle enormously thickened. Kidneys atrophied (gouty).

Lobelia, henbane and ether (3 days). Then carbonate of ammonia and citrate of iron and quinine. Turpentine stupes. J. L. to præcordia. Brandy 6 oz.

*Aneurism of Aorta.—J. H., æt. 31, Hawker of playbills. Adm. July 16. *Died* July 20. In H. 4 days. Dr. Johnson, vol. 19, p. 7. Intemperate. Has to run much. Suddenly lost voice 7 weeks ago. Dysphagia 8 days. Cough dyspnœa, and increased hoarseness, 5 days. On adm. voice husky. Breathing

stridulous. No bruit. Laryngoscopic examination showed larynx healthy. Pain at lower end of sternum; 2 days later slight hœmoptysis; 2 days later sudden faintness, vomiting of blood twice, and death. P.M.—An aneurism of descending arch, as large as an orange, opening into œsophagus by a valvular aperture the size of a florin. Recurrent laryngeal compressed by it. Stomach full of blood.

Quinine. Gallic acid.

Aneurism of Aorta.—Mary A. O., æt. 32, married. Brought in dead on Sept. 14. Dr. Johnson. Fell down in street. Had been dyspeptic 4 or 5 months. P.M.—A small aneurism of arch of aorta, at junction of ascending and transverse portions, the sac between the aorta and pulmonary artery. Hemorrhage into pericardium through a triangular aperture in left side of aneurism.

Cardiac Disease.—M. A. M., æt. 29, Needlewoman. Adm. Nov. 16. Disch. Nov. 28. *Relieved.* Dr. Johnson, vol. 17, p. 153. Intemperate. Ill 2 years. Palpitation and stabbing pain in præcordial region. Has spat blood. Cardiac dulness increased. Systolic bruit loudest at base. P. 78 feeble. Dulness and wavy expiration at left apex.

Iron and quinine.

Cardiac Disease.—Emma B., æt. 27, married. Adm. Oct. 3. *Died* on Oct. 3. Dr. Garrod, vol. 3, p. 54. Acute rheumatism 6 months ago. Dropsy 3 months. On adm. almost moribund, face blue; great dyspnœa; cardiac dulness increased; double bruit at base; urine ¼th albumen. P.M.—Fluid in pleuræ pericardium and peritoneum. Heart large; mitral valve roughened with fibrinous deposits; warty growths on aortic valves. Lungs œdematous. Kidneys wasted.

Liq. ammon. Acetatis, acetate of potass, and tincture of belladonna.

Cardiac Disease.—Joseph H., æt. 36. Adm. Dec. 9. *Died* Dec. 9. Dr. Johnson, vol. 19, p. 189. Very sallow. Systolic bruit over apex of heart. Died of syncope a few minutes after adm. P.M.—General emphysema of lungs. Heart large; left ventricle thickened and dilated; mitral valve incompetent; aortic orifice and arch much dilated. Kidneys wasted.

Cardiac Disease.—J. W., æt. 32, Porter at Gas Works. Adm. Dec. 8. Disch. Dec. 19. In H. 11 days. Dr. Beale, vol. 8, p. 10. Drinks freely. Never had rheum. Winter cough 5 years. Jaundice 15 months ago. Palpitation and dyspnœa 6 months. Slight œdema of legs; pain over heart; cough and dyspnœa. Cardiac dulness increased; pulsation in scrobiculus; action irregular. Rhonchus and sibulus over lungs. P. 102. Pain, tenderness, and enlargement of liver; some yellowness of conjuctivæ; urine albuminous. Discharged at his own request.

Carbonate of ammonia, chloric ether and senega. Jalap and scammony. Back dry cupped. Brandy 6 oz.

Cardiac Disease.—Mary R., æt. 34, Street Hawker. Adm. Oct. 27. Disch Dec. 2. In H. 36 days. *Relieved.* Dr. Johnson, vol. 18, p 69. Never had rheumatism Hæmoptysis 6 months ago. Cough and slight hæmoptysis since. Loss of flesh and strength 3 months. Anæmic. Pulse very feeble. Rough systolic bruit and thrill at union of 2nd left rib with cartilage; soft double bruit at base. Dulness and large crepitation at apex of left lung. Fingers clubbed.

Sesquichloride of iron. Morphia at night.

Cardiac Disease.—W. B., æt 21, Labourer. Adm. July 17. Disch. Aug. 29. In H. 43 days *Relieved.* Dr. Johnson, vol. 19, p. 12. Acute rheumatism 8 years ago. Palpitation 2 years. Three weeks ago violent palpitation, pain in chest dyspnœa. and hæmoptysis (½ pt.). Has spat blood since. On adm. Florid. Has orthopnœa. R. 28 easily hurried. Dulness and fine crepitation over base of right lung. Heart's apex between 6th and 7th ribs. 1¼ in. to left of nipple; strong impulse here and in scrobiculus. Systolic bruit loudest at apex, and loud diastolic bruit at base. P. full, collapsing, 120. Expectorates much dark blood.

Had a second attack of hæmoptysis on day after adm. Still a trace of blood in expectoration when discharged at own request.

Dilute sulphuric acid, chloric ether, opium, and digitalis. Turpentine. Quinine and sesquichloride of iron. Dry cupping to chest.

Cardiac Disease.—J. B., æt. 35, Warehouseman. Adm. Aug. 5. Removed by relatives Aug. 9. In H. 4 days. *Relieved.* Dr. Garrod, vol. 1, p. 195. Acute rheum. 11 years ago. Cough and loss of strength 5 months. Pain in chest and palpitation 4 months. Œdema of legs and great dyspnœa 3 weeks. Very anœmic. Cardiac region extremely tender. Impulse heaving. Systolic bruit at apex and diastolic bruit at base. P. feeble and irregular, 108. R. 34. Urine very albuminous.

Dilute hydrocyanide acid, sulphuric ether and digitalis.

Cardiac Disease.—J. M. M., æt. 54, Porter. Adm. June 23. Disch. July 15. In H. 22 days. *Relieved.* Dr. Garrod, vol. 1, p. 153. Intemperate. Never had rheumatism. Cough and expectoration 5 months. Loss of appetite 3 months. Shortness of breath and palpitation 2 months. Copious rusty expectoration 3 days. Very anœmic. Legs slightly œdematous. Impulse of heart feeble. Double bruit over base. P. 88, collapsing, and locomotive. Slight dulness below spine of right scapula. Crepitation at base. Abundant bright rusty expectoration. Gout in left great toe 6 days later.

Carbonate of ammonia and ammonic citrate of iron.

Cardiac Disease.—Caroline A., æt. 53. Bootbinder. Adm. July 8. Disch. July 24. In H. 17 days. *Relieved.* Dr. Beale, vol. 6, p. 226. Never had rheumatism. Cough 20 years. Shortness of breath and palpitation 2 years. Weakness and emaciation 3 weeks. Heart's action irregular. Apex to left of left nipple. Cardiac dulness enlarged. Pulsation in scrobiculus. Systolic bruit at apex. P. 120, small, and irregular. Sibilus and crepitation over chest. Cough and scanty expectoration. Appetite bad. Sleeps badly.

Digitalis and chloric ether. Sal volatile and chloric ether.

Cardiac Dropsy.—J. T., æt. 53, Butcher. Adm. June 20. *Died* on July 16. In H. 26 days. Dr. Garrod, vol. 1, p. 148. Had acute rheumatism when 7 years old. Winter cough 10 years: worse 3 months. Legs dropsical 1 month: inflamed 5 days. Has marked arcus senilus: conjunctivæ yellow. Heart's sounds distant, action irregular. P. 116. No bruit. Crepitation over bases of lungs. Dulness at right base. Urine free from albumen throughout. Sloughing of inner side of right leg. Dyspnœa increased. Death from exhaustion.

Bark and carbonate of ammonia (7 days). Then carbonate of ammonia, citrate of iron, and decoction of senega. Turpentine stupes.

Cardiac Dropsy.—R. S., æt. 50, Barrister's clerk. Adm. Nov. 5. *Died* on Dec. 5. In H. 30 days. Dr. Johnson, vol. 19, p. 142. Never had rheumatism. Intemperate. Prev. ill. 5 weeks. On adm. Pallid. Legs, thighs, and scrotum œdematous. Sort systolic bruit at apex of heart. Afterwards much ascites, hæmaturia delirium and death. P.M. Recent lymph at lower and back part of right pleura. Heart small. Tricuspid valve thickened. Kidneys pale.

Iron, quinine, and various diuretics. Hydragogue purgatives.

Cardiac Dropsy.—Susan B., æt. 40, Married. Adm. Sept. 14. *Died* on Nov. 28. In H. 75 days. Dr. Johnson, vol. 19, p. 59. Acute rheumatism 8 years ago. Legs œdematous about 2 months, had been much swelled before confinement 3 months ago. Slight ascites. Loud systolic bruit over heart's apex. P. 80. Dulness and crepitation below scapulæ. Albumen and waxy casts in urine. 60 days later much crepitation over lungs, dropsy much increased, legs punctured (twice). Slight improvement. Gangrene of legs. Death. P.M. Some patches of pulmonary apoplexy. Cragula in pulmonary artery. Mitral valve much thickened. Tricuspid orifice dilated. Kidneys wasted. Liver slightly cirrhosed. Many gall stones.

Quinine, iron, and stimulants. Legs punctured. Loins dry cupped.

*Cardiac Dropsy.—B. S., æt. 29, Street Hawker. Adm. Nov. 18. *Died* Dec. 18. In H. 30 days. Dr. Johnson, vol. 17, p. 155. Shortness of breath, cough, and palpitation 4 months. Œdema of legs 1 week. Never had rheumatism. On adm. cardiac dulness 4 in. transversely. Apex to the left; impulse strong. Systolic bruit at apex loudest to left of ensiform cartilage. Soft diastolic bruit over base. P. 110, small. Cough, dyspnœa, and viscid expectoration. Rhonchus and crepitus over back of lungs, dulness at bases. Liver tender. Urine slightly albuminous. 11 days later severe pain about right nipple; great dyspnœa. 2 days later pleuritic rub below nipple. 1 day later dulness below angle of right scapula. 13 days later dulness below 4th rib in front and spine of scapula on right side; crepitation behind; sputa rusty; urgent dyspnœa. 2 days later much pain about nipple; more dyspnœa; delirium; death. P.M. General anasarca. Right pleura full of fluid; lymph on diaphragmatic and pericardial layers. Right lung carnefied: its lower lobe contained patches of circumscribed pulmonary apoplexy. Heart 27 oz. Aortic arch atheromatous and dilated. Pouch over one semilunar valve. Ventricles and auric-ventricular orifices much dilated. Margin of tricuspid valve thickened.

Ammonia and ether. Brandy. Henbane and Indian hemp. Purgatives. Chest dry cupped.

Cardiac Dropsy.—M. A. B., æt. 45, Nurse. Adm. Oct. 12. *Died* on Oct. 17. In H. 5 days. Dr. Garrod, vol. 3, p. 70. Palpitation and œdema of legs on and off 2 years. Hæmatemesis 18 months ago. On adm., anasarca, slight ascites. Systolic bruit loudest at apex. Crepitus at bases of lungs, liver much enlarged and tender, conjunctiva yellow. P. 120, R. 40. Appetite bad. 3 days later delirious. Died of syncope. P.M. Heart fatty. Liver large and fatty.

Dilute muriatic acid, chloric ether, and bark. Carbonate of ammonia. Brandy.

*Cardiac Dropsy.—W. H., æt. 56, Millwright. Adm. Sept. 19. *Died* on Nov. 1. In H. 43 days. Dr. Johnson, vol. 19, p. 72. Very intemperate. Cholera 28 years ago. Typhus 23 years ago. Cough and hoarseness 2 years. Palpitation 12 months. Dropsy 14 days. On adm. conjunctiva slightly yellow; cough raucous. Physical signs of pulmonary emphysema. Musical bruit at apex, systolic, and prolonged into diastole; rough bruit at base. Dulness over base of right lung; bronchial br. about angle of scapula. Hæmoptysis 4 days later. Dulness over left base 2 days later. Urine slightly albuminous 24 days later. P.M. Lungs gorged and emphysematous. Heart 15 oz. Mitral and tricuspid orifices dilated. Trachea compressed by an almost aneurismal dilatation of aortic arch.

Stimulants. Diuretics. Hydragoque purgatives. Brandy to 15 oz. Chlorodyne. Iron and bark. Legs punctured twice.

*Cardiac Dropsy.—A. V., æt. 52, Needlewoman. Adm. June 20. Disch. Sept. 30. In H. 102 days. *Recovery.* Dr. Garrod, vol. 2, p. 190. Had scarlet fever 4 times. Acute rheum. 22 years ago. Subject to bronchitis 10 years. Typhoid fever 1½ years ago. Cough and shortness of breath 3 months. Palpitation 2 months. Dropsy of legs 14 days. On adm. considerable œdema of legs. Slight ascites. Dulness below spine of right scapula and some crepitation; elsewhere sibilus. Heart irregular; soft systolic bruit at apex. P. 80, R. 28. Urine slightly albuminous. 31 days later urine 100 oz. daily; dropsy became much less, and ultimately disappeared.

Carbonate of ammonia and iodide of potassium, and bitartrate of potass and jalap powders. Digitalis. Afterwards iron columba and digitalis.

Cardiac Dropsy.—R. B., æt. 32, Traveller. Adm. June 29. Disch. July 18. In H. 19 days. *Much relieved.* Dr. Garrod, vol. 2, p. 211. Intemperate. Never had rheum. Typhus 18 years ago. Palpitation, headache, and vomiting occasionally for 12 months. Weakness, œdema of legs, cough, and constipated bowels, 5 weeks. On adm. very pallid, legs slightly œdematous. Loud systolic bruit at apex audible all over chest. Dulness and diminished vocal vibration below spine of right scapula. Urine ¼th albumen.

Carbonate of ammonia and digitalis. Bitartrate of potass and jalap.

Cardiac Dropsy.—E. T., æt. 18, Housemaid. Adm. Nov. 21. Disch. Dec. 12. In H. 21 days. *Recovery.* Dr. Johnson, vol. 18, p. 111. Acute rheumatism 10 years ago. Palpitation and shortness of breath on exertion 6 weeks. Œdema of legs 7 days. Heart's impulse strong. Systolic bruit at base, and a softer one at apex. P. 112, feeble. Is anœmic.

Sesquichloride of iron and quinine. Compound jalap powder.

Endocarditis.—E. S., æt. 39, Hospital Nurse. Adm. Nov. 11. Disch. Nov. 26. In H. 15 days. *Recovery.* Dr. Beale, vol. 5, p. 353. Prev. ill. 1 day. Sudden præcordial pain and symptoms of angina. Feverishness. Rough systolic bruit over base of heart faintly audible 12 days later.

Liquor ammoniæ acetatis, liq. cinchonæ and sp. ammon. arom. (6 *days*). *Then dilute muriatic acid and decoction of bark. Morphia at night.*

Angina Pectoris.—G. A., æt. 62, Tailor. Adm. Aug. 27. *Died* on Aug. 27. Dr. Garrod, vol. 2, p. 268. Picked up in street by police. On adm. face pale, lips blue, severe pain at heart pulse imperceptible. Left leg and thigh red, swelled, and painful Heart's sounds feeble; 2nd inaudible at apex; faint basic diastolic bruit. Has drunk hard. Cardiac pain and uneasiness some years. At 7 p.m. had an attack of dyspnœa, and died in a few minutes. P.M.—Lungs gorged. Heart flabby, 11¾ oz. Aortic valves atheromatous and incompetent. Coronary arteries ossified. Liver and kidneys large.

Aromatic spirits of ammonia, ether, and tinct. card. co.

Acute Bronchitis.—W. T., æt. 66, Commissionaire. Adm. Sept. 21. Disch. Oct. 10. In H. 19 days. *Recovery.* Dr. Johnson, vol. 17, p. 93. Cough 3 months. Dyspnœa 1 week. On adm. headache, muscular cramps, cough, and mucous expectoration. Rhonchus and sibilus over chest, Tongue furred. P. 100. Highest resp. 32.

Chloric ether and carbonate of ammonia. Chlorodyne. Turpentine stupes.

Acute Bronchitis.—S. G., æt, 58, Charwoman. Adm. Oct. 8. *Died* Oct. 9. Dr. Garrod. Had rheumatism. Breath short for some years. On adm. very weak, pulse small, lips purple. Bedsore over sacrum. Rhonchus and large crepitation over lungs. P.M.—Aortic valves thickened. Mucous membrane of small bronchial tubes intensely inflamed.

Ammonia and chloric ether. Brandy 8 *oz.*

Acute Bronchitis.—C. W., Hospital Nurse. Adm. Dec. 22. Disch. Dec. 31. In H. 9 days. *Recovery.* Dr. Beale. Prev. ill. 1 day. On 3rd day P. 120, R. 36. Rhonchus and sibilus over back of chest. Yellow puriform expectoration.

Aromatic spirits of ammonia, chloric ether, squills, and bark.

Bronchitis—Dropsy.—Selina S., æt. 50. Adm. Oct. 29. Disch. Nov. 11. In H. 13 days. *Recovery.* Dr. Beale, vol. 5, p. 340. Ill 9 weeks. Cough and frothy mucous expectoration. Dyspnœa; œdema of legs. Dulness and crepitation at posterior bases; rhonchus and sibilus elsewhere. P. 80, R. 48. Area of cardiac dulness increased, and first sound prolonged.

Carbonate of ammonia, chloric ether, and senega. Scammony and jalap powder. Turpentine stupes.

Bronchitis.—A. C., æt. 35, Tailoress. Adm. Oct. 30. Disch. Nov. 21. In H. 22 days. *Recovery.* Dr. Garrod, vol. 3, p. 126. Sore throat and pain in swallowing 1 month. Aphonia 1 week. Crepitus at right posterior base. Sputum scanty. Redness of left anterior faucial pillar.

Liq. ammon. acetatis, carbonate of ammonia, and ipecacuanha (13 *days*). *Bark and nitric acid.*

Bronchitis.—A. B., æt. 37, Needlewoman. Adm. Sept. 23. Disch. Oct. 21. In H. 28 days. *Recovery.* Dr. Beale, vol. 6, p. 274. In K. C. H. during Jan., 1863, for 2nd attack of rheum. Prev. ill. 14 days. Œdema of legs. Pain, redness, and swelling of left ankle. Cough and free expectoration. Crepitation over left lung and base of right, elsewhere rhonchus. Systolic bruit loudest at apex. P. 96, R. 40. Slight hepatic enlargement. Tongue furred. Epigastrium tender. Appetite bad.

Sesquichloride of iron, chloric ether, and calumba. Then sp. ammon. arom. and chloric ether.

*Bronchitis—Pleurisy.—Mary H., æt. 20, Servant. Adm. Aug. 6. Disch. Sept. 19. In H. 45 days. *Recovery.* Dr. Johnson. Prev. ill. 6 days. On 7th day cough, pain in chest, brown tongue, bad appetite, crepitation at bases, rhonchus and sibilus over right lung. On 11th day P. 120, more crepitation. On 13th day dulness and feeble breathing over left lower lobe. Tubular breathing at left scapular angle on 15th day. On 18th day brown dry tongue, purged; sores on sacrum, delirious, P. 124, R. 44, urine passed under about this time. Convalescent on 31st day.

Liquor ammon. acetat. (5 days). Sulphate and carbonate of magnesia and chloric ether (4 days). Liq. ammon. acetat., chloric ether, and sal volatile. Quinine and acid on 22nd day. Brandy to 24 oz. Turpentine stupes.

Chronic Bronchitis—Pleurisy.—E. F., æt. 36, Charwoman. Adm. Aug. 5. Disch. Sept. 9. In H. 35 days. *Recovery.* Dr. Garrod, vol. 1, p. 197. Cough every winter. Prev. ill. 1 month. Shivering, dyspnœa, pain in left side. On adm. P. 108, R. 34, expectoration muco-purulent. Dulness diminished, vocal vibration and feeble breathing below left scapular angle; large crepitation over right sup. spinous fossa; elsewhere rhonchus and sibilus. 3 days later large crep. below scapular spines; left base dull. 5 days later br. br. and bronchophony over left lower lobe behind. 9 days later a patch of br. br. at left scapular angle; elsewhere rhonchus and crepitation.

Carbonate of ammonia (4 days). Chloric ether and aromatic spts. of ammonia (11 days). Senega and carbonate of ammonia. Turpentine stupes.

Bronchitis and Emphysema.—Elizabeth S., æt. 53. Adm. May 20. *Died* on July 6. In H. 47 days. Dr. Johnson, vol. 15, p. 199. Winter cough 8 years. Prev. ill. 3 weeks. Face extremely livid and congested. Legs slightly œdematous. Mucous rates over chest. Scanty viscid expectoration. P. 120, R. 48. Urine albuminous about 14th day after adm. Legs punctured 14 days later. Copious drain at first, afterwards intense inflammation. Death from exhaustion.

Carbonate of ammonia, chloric ether, and senega. Diuretics. Elaterium. Chlorodyne. Dry cupping. Cucurb. cruent. ad ʒiv. on 6th day after adm.

Bronchitis and Emphysema.—J. K., æt. 36, Hatter. Re-adm. Oct. 10. Disch. Nov. 21. In H. 42 days. *Much relieved.* Dr. Beale, vol. 6, p. 302. O. P. since disch. on May 14. Worse about a week; face livid; anasarca; slight ascites; much dyspnœa. Sibilus over lungs above, crepitation at bases, slight dulness at left base. P. 100, R. 28. Systolic bruit over heart. Urine albuminous.

Carbonate of ammonia, chloric ether, and senega. Dilute muriatic acid, chloric ether, and squills. Compound jalap powder. Brandy.

*Bronchitis and Emphysema.—J. H., 49, Clerk. Adm. Nov. 27. *Died* on Dec. 18. In H. 21 days. Dr Garrod, vol. 4, p. 28. Intemperate. Ill 4 months. Pallid and anasarcous; slight ascites; rhonchus and sibilus over lungs; dry cough; P. 104. 4 days later more or less delirious; motions and urine passed under him. On 18th day coarse crepitus all over lungs. P.M.—Much pulmonary emphysema. Left ventricle dilated and hypertrophied. Kidneys wasted and granular. Old clot in left corpus striatum.

Squills, ammonia, and decoction of bark. Iron and quinine. Brandy, 12 *oz.*

Bronchitis and Emphysema.—C. W., æt. 52, Cook. Adm. Sept. 28. Disch. Oct. 17. In H. 19 days. *Recovery.* Dr. Johnson, vol. 17, p. 118. Subject to winter cough. Prev. ill. 5 weeks. Face dusky. Legs œdematous. Slight dry cough. P. 96, R. 28. Rhonchus and sibilus all over chest. Pulsation in scrobiculus.

Chloric ether and carbonate of ammonia. Turpentine stupes.

Bronchitis and Emphysema.—Mary K., æt. 50. Adm. Oct. 14. Disch. Oct. 24. In H. 10 days. *Relieved.* Dr. Johnson, vol. 17, p. 130. Winter cough 12 years. Prev. ill. 6 weeks. Lips blue; legs œdematous; cough and mucous expectoration; universal coarse crepitation.

Carbonate of ammonia, squills, and decoction of broom tops.

Bronchitis and Emphysema.—M. B., æt. 35, Waterman. Adm. Sept. 16. Disch. Oct. 13. In H. 27 days. *Recovery.* Dr. Beale, vol. 6, p. 250. Intemperate. Winter cough 2 years. Has spat blood. Prev. ill. 14 days. Cough and abundant expectoration. Rhonchus and sibilus all over chest. Slight dulness, feeble breathing, and diminished vocal vibration at left base. Tongue dry and furred. P. 108, R. 32.

Carbonate of ammonia and senega. Then carbonate of ammonia, liq. ammon. acetatis and decoct. cinchonæ. Turpentine stupes.

Bronchitis and Emphysema.—Maria S., æt. 38. Adm. Sept. 28. Disch. Oct. 3. In H. 11 days. *Much relieved.* Dr. Garrod, vol. 3, p. 39. Cough and short breath 10 years. Acute rheum, 2 years ago. Face blue, legs œdematous, cardiac dulness increased, rhonchus sibilus and crepitation over lungs, puriform expectoration. P. 96, R. 36. Tenderness over liver.

Carbonate of ammonia, decoction of senega and sp. eth. Nit. Elaterium. Brandy 2 *oz. Turpentine stupes.*

Chronic Bronchitis.—Catherine S., æt. 36. Adm. June 10. Disch. Aug. 12. In H. 63 days. *Unrelieved.* Dr. Beale, vol. 6, p. 186. Ill about 6½ months. Lately in St. George's. Now much sibilus over chest. Expectoration puriform. P. 60.

Ammonia and chloric ether. Sedatives.

Catarrh.—C. M., æt. 21, Frame Maker. Adm. July 13. Disch. July 24. In H. 11 days. *Recovery.* Dr. Johnson, vol. 17, p. 51. Ill three weeks. Much debility.

Quinine and sesquichloride of iron.

Catarrh.—E. B., æt. 15, Nurserymaid. Adm. July 3. Disch. July 8. In H. 5 days. *Recovery.* Dr. Beale, vol. 6, p. 220.

Liq. ammon. acetatis, sp. ammon. arom., and chloric ether.

Pneumonia.—P. P., æt. 36. Adm. June 24. Disch. July 15. In H. 21 days. *Recovery.* Dr. Johnson, vol. 15, p. 255. Prev. ill. 4 days. Got wet through. Then shivering, cough, dyspnœa and pain in right side. Expectoration rusty on 3rd day (9 days). On adm. skin hot, tongue dry and brown. Dulness and increased vocal vibration over back and upper front of right lung. Bronchial breathing (1 day), over dull space above, small crepn. below. Then returning crepn. Trace of albumen in urine; chlorides scanty. Highest pulse 116, highest resp. 48.

Chloric ether, carbonate of ammonia, and liq. ammonia acetatis (5 *days*). *Then quinine. Brandy* 8 *oz. Turpentine stupes.*

Pneumonia.—Edward G., æt. 7. Adm. Nov. 19. Disch. Dec. 29. In H. 40 days. *Recovery.* Dr. Johnson, vol. 17, p. 157. Ill 3 days. Vomiting. Pain in

right side. Breath short. Tongue furred. P. 144. Acute tenderness of right chest (5 days). Some dulness at right base. Br. br. about scapular angle. On 8th day tubular br. over upper lobe. On 10th day br. br. over scapula. On 12th loud br. br. over right apex. P. 120, R. 30. Br. everywhere vesicular on 33rd day.

Liq. ammon. acetatis, ipecacuanha, sp. eth. nit., and liq. morph. mur. (8 *days*). *Then quinine. Wine* 8 *oz. Cod liver oil. Sinapism to back.*

Pneumonia.—Harry T., æt. 10. Adm. June 15. Disch. July 11. In H. 26 days. *Recovery.* Dr. Garrod, vol. 2, p. 182. Ill 1 day, with shivering, cough, loss of appetite and pain in left side. Now flushed, left chest painful and tender on pressure (4 days). Dulness, loud bronchial br., and bronchophony below spine of left scapula (14 days). No expectoration. Highest pulse 144, highest resp. 84.

Chloric ether, carbonate of ammonia and liquor ammoniæ acetatis. Afterwards quinine.

Pneumonia-Phlegmasia dolens.—C. L., æt. 24, Waiter. Adm. Aug. 7. Disch. Sept. 26. In H. 50 days. *Recovery.* Dr. Beale, vol. 5, p. 256. Slight purpura 3 weeks. Phlegmasia dolens of right leg 2 weeks. Pain in right chest on day after adm., hæmoptysis next day; 2 days later dulness over lower lobes of lungs, small crep. over right, large crep. over left, sputa bloody, P. 100, R. 36; 3 days later loud pleuritic rub over right lower lobe; 4 days later right leg larger, fresh petechiæ; 5 days later phleg. dolens of left leg.

Liq. cinchonæ and citrate of potass. Dilute muriatic acid and bark. Belladonna ointment to legs.

Pneumonia.—J. S., æt. 38, Sign Writer. Adm. July 2. Disch. Sept. 19. In H. 79 days. *Recovery.* Dr. Beale, vol. 5, p. 216. Prev. ill. 5 days. Slight hæmoptysis, recurring on morning of adm. Pain in left side. Now P. 72, slight dulness and feeble br. over whole left lung. Four days later, increased dulness and diminished vocal vibration over left side, behind, bronchial br. below angle of scapula, expectoration viscid and rusty. Next day large crepn., mixed with br. br. below scap. angle. P. rose to 106. On 67th day friction sound over left base. On 75th day br. vesicular, rub still audible.

Gallic acid and opium. Then sal volatile, chloric, ether and liq. ammon. acetatis. Nitric acid and bark. Compound iodine and mercurial ointment applied over lungs.

Pneumonia.—E. W., æt. 20, Salesman. Adm. Oct. 23. Disch. Nov. 16. In H. 24 days. *Recovery.* Dr. Beale, vol. 5, p. 326. Prev. ill. 5 days. Shivering, cough, loss of appetite, slight hæmoptysis. Dulness, fine crepitation and increased vocal vibration over right lower lobe below scapular angle, up to spine 4 days later; sputa rusty at first; now viscid and colourless. Highest pulse 84, highest resp. 48.

Sp. ammon. arom., and liquor ammoniæ acetatis. Dilute muriatic acid and quassia. Brandy 6 *oz. Turpentine stupes. E. L. to right side.*

Pneumonia.—Mary A. C., æt. 14. Adm. July 14. Disch. Aug. 12. In H. 29 days. *Recovery.* Dr. Johnson, vol. 18, p. 1. Ill 2 days. Shivering, headache, thirst, anorexia. Tongue furred. Dry cough. Much pain and tenderness on pressure, slight dulness, and harsh bronchial expiration under right clavicle. Some dulness at right base. P. 124. R. 64.

Chloric ether and liquor ammon. acetatis. Then quinine and iron. Aperients.

Pleuro-Pneumonia.—H. W., æt. 19, Compositor. Adm. Sept. 26. Disch. Nov. 7. In H. 42 days. *Recovery.* Dr. Johnson, vol. 19, p. 88. Prev. ill. 6 days. Pain in left side, dyspnœa, cough, hæmoptysis. On adm. dulness, bronchial

breathing, œgophony, friction sound, and diminished vocal vibration, below left scapula. Expectoration viscid and rusty.

Liquor ammonia acetatis (9 *days*). *Then sesquichloride of iron and quinine. E. L. to left side on* 16*th day.*

Pleurisy.—G. E., æt. 37, Carpenter. Adm. July 1. Disch. Sept. 14. In H. 75 days. *Recovery.* Dr. Johnson, vol. 16, p. 263. Prev. ill. 6 weeks. Began with pain in right side. On adm. P. 100, R. 32, right side dull all over behind, and below 3rd rib in front (up to clavicle on 7th day after adm.). Absence of breathing in front; œgophony and faint tubular breathing below angle of scapula behind; vocal vibration diminished. 42 days later breathing everywhere vesicular.

Iodide of potassium, chloric ether and bark (14 *days*). *Then quinine and acid. Ol. Morrhuæ. Iodine paint to right side.*

Pleurisy.—M. G., æt. 35, Pavior. Adm. June 11. Disch. July 1. In H. 20 days. *Relieved.* Dr. Beale, vol. 6, p. 182. Pain in left side 3 weeks. On adm. dulness, diminished vocal vibration, and absence of breathing below 3rd rib in front, and spine of left scapula. Pleuritic rub above 3rd rib. P. 120, R. 38. 15 days later loud friction sound, and feeble breathing all over left side.

Liq. ammoniæ acetatis, carbonate of ammonia, chloric ether and broom tops.

Pleurisy.—Sam. B., æt. 30, Singer. Adm. Nov. 13. Disch. Dec. 15. In H. 32 days. *Recovery.* Dr. Johnson, vol. 19, p. 152. Prev. ill. 3 days. Stabbing pain in left side. Dulness with absence of breathing, and vocal vibration below 3rd rib in front and angle of left scapula. Pleuritic rub below left nipple. Œgophony. Sputa, viscid, untinged. On 11th day coarse crep. at left post. base (21 days). Much pain in side during recovery.

Aromatic spts. of ammonia, liq. ammon. acetatis, and bark (4 *days*). *Then quinine. Iodine paint to side.*

Pleurisy.—W. S., æt. 28, Carrier. Adm. Oct. 16. Disch. Dec. 9. In H. 54 days. *Recovery.* Dr. Johnson, vol. 18, p. 43. Prev. ill. 21 days. Dyspnœa and pain in right side Œdema of legs 15 days later. On adm. dulness below 5th rib, in front, and right scapular spine. Friction sound near nipple. Breathing distant above, bronchial at angle, absent at base. Sputum clear. P. 78, R. 30. 31 days later flattening of right chest and sinking of right shoulder.

Liq. ammoniæ acetatis, chloric ether and aromatic spts. of ammonia.

Pleurisy.—H. S., æt. 23, Labourer. Adm. Oct. 1. Disch. Nov. 7. In H. 37 days. *Recovery.* Dr. Beale, vol. 5, p. 308. Prev. ill. 12 days. Pain in right side, dry cough, dyspnœa. Dulness, absence of breathing, and vocal vibration below upper margin of right scapula, and below nipple. P. 96, R. 40. Friction sound below right scapular angle on 17th day (20 days), and bronchial breathing at scapula angle about 24th day.

Liquor ammoniæ acetatis, sp. ammon. arom., and chloric ether. E. L. to right side on 31*st day.*

Pleurisy.—Mary H., æt. 20, Servant. Adm. June 24. Disch. July 18. In H. 24 days. *Recovery.* Dr. Johnson, vol. 15, p. 261. Prev. ill. 3 weeks. Pain in left side, dry cough. Dulness below 4th rib, in front, and centre of left scapula. Breathing absent at base, feeble elsewhere. Slight œgophony. Five days after adm. pleuritic rub at base, audible till discharge.

Liquor ammoniæ acetatis and chloric ether (14 *days*). *Then quinine and iron. Aperients.*

Pleurisy.—Sarah B., æt. 35, Servant. Adm. July 6. Disch. Aug. 12. In H. 37 days. *Recovery.* Dr. Beale, vol. 5, p. 220. Prev. ill. 3 days. Abdominal and interscapular pain, vomiting, shivering, and loss of appetite. On adm. tongue furred, P. 120, R. 24. Next day pleuritic rub (22 days) at right scapular angle. Later, dulness and absence of breathing at base. Liver enlarged.

Liq. ammoniæ acetatis, chloric ether and aromatic spts. of ammonia. Then

dilute nitric acid and bark. Taraxacum. Purgatives. Hydrochloric acid lotion.

Pleurisy.—F. N., æt. 20, Sempstress. Adm. June 17. Disch. July 1. In H. 14 days. *Recovery.* Dr. Beale, vol. 6, p. 192. Prev. ill. 7 days. Pain in right side, dry cough, thirst, loss of appetite. On adm. P. 108, R. 40. Slight dulness, feeble breathing, and diminished vocal vibration below right scapular angle; friction sound two days later.

Liquor ammon. acetatis (5 days). Then iron and quinine. E. L. to side.

Pleurisy.—Eliza B., æt. 20, Servant. Adm. Nov. 14. Disch. Dec. 2. In H. 11 days. *Recovery.* Dr. Beale, vol. 6, p. 323. Pain in right side 6 days. On adm. friction sound near angle of right scapula (5 days). P. 92. Probably tubercular.

Liq. ammon. acetatis and sal volatile (10 days). Quinine. Brandy. Aperients.

Empyema.—Edwin H., æt. 13. Adm. Aug. 24. Disch. Oct. 7. In H. 44 days. *Relieved.* Dr. Garrod, vol. 2, p. 261. Prev. ill. 8 months. At first febrile symptoms and pain in left side. Five months later a tender red tumour between left 4th and 5th ribs 2 in. below nipple, and 1½ in. to left of sternum; opened 6 weeks ago. Now pallid, left shoulder sunk, side flattened, girth at nipple 2 in. less than right side, about ʒiii. pus daily from opening. Left side dull except above 3rd rib in front. Vocal tremor absent. Breathing vesicular to mid scapula, below this to base bronchial with ægophony, harsh and crackling under clavicle. P. 88. Skin hot and dry. Discharge fell to ʒ½ daily.

Nitric acid and bark. Afterwards cod liver oil and syrup of iodide of iron. Left side tightly strapped below opening.

Phthisis.—J. E., æt. 81. Adm. June 16. *Died* on July 31. In H. 45 days. Dr. Garrod, vol. 2, p. 174. Cough 2 years. Worse 4 days ago. Right upper lobe dull on percussion, cavernous breathing, gurgling and pectoriloquy under clavicle, pectoriloquy at post. apex. P. 96. P.M. Right lung adherent, 1 pint fluid in base of pleura. Right lung stuffed with tubercles; vomica in post. apex. Slight tubercular deposit in left apex.

Carbonate of ammonia and bark. Cod liver oil. Dilute sulphuric acid and bark.

Phthisis.—T. C., æt. 47, Silver Polisher. Adm. Nov. 10. *Died* on Nov. 15. In H. 5 days. Dr. Johnson, vol. 19, p. 148. Cough 3 winters. Loss of flesh 6 months. Hoarse 2½ months. Dulness, amphoric breathing, and cracked pot sound under left clavicle; dulness, br. br., and crep. over left back. Tubular br. and crep. under right clavicle. P. 120. P.M.—Large cavern in left upper lobe. Much tubercle elsewhere in lungs. Liver large.

Sp. ammon. arom., hyoscyamus and bark. Syrup of iodide of iron and cod liver oil.

Phthisis.—J. M., æt. 27, Coachman. Adm. Sept. 20. *Died* on Oct. 30. In H. 40 days. Dr. Garrod, vol. 3, p. 21. Hereditary. Cough and loss of flesh, 5 months. Diarrhœa. Hæmoptysis at times, 6 weeks. 3 pts. blood on day before adm. Dulness and fine crep. at left apex; gurgling and tubular br. under right clavicle; crep. at post. apex; slight dulness and fine crep. over right back; P. 120, R. 40. Sputa nummular streaked with blood; 1½ pints daily twenty days later.

Dilute phosphoric acid, glycerine, morphia, and gentian. Syrup of iodide of iron and cod liver oil. Liq. ammon. acet., digitalis, and bark. I. P. to right chest.

Phthisis.—W. B., æt. 49, Compositor. Adm. July 27. Disch. Aug. 8. In H. 12 days. *Unrelieved.* Dr. Johnson, vol. 19, p. 34. Always subject to cough and expectoration. Very anæmic. Dulness under left clavicle; large crep. to nipple;

crep. and prolonged expectoration at left post. apex ; crep. under right clavicle. Puriform sputa. P. 100. Night sweats. Liver large and tender.

Cod liver oil. Dilute muriatic acid, chloric ether, and quinine.

Phthisis.—Cath. C., æt. 13. Adm. July 29. Disch. Aug. 5. In H. 7 days. Dr. Beale. Cough and loss of flesh 5 months. Hæmoptysis on evening of adm. and 4 days ago. Slight dulness over left upper lobe ; loose crep. and tubular br. under clavicle ; smaller crep. below. Harsh br. and crackling under right clavicle. Sputa nummular.

T. opii, dilute sulphuric acid, and chloric ether. Cod liver oil. E. L. under left clavicle.

Phthisis.—Robert E., æt. 24. Adm. Sept. 26. Disch. Oct. 31. In H. 35 days. *Very much relieved.* Dr. Beale, vol. 5, p. 298. Had phthisis 6 years. Worse 3 months. Vomited blood 2 years ago. Frequent hæmoptysis. Left lung dull back and front. Gurgling tubular br. and pectoriloquy under clavicle ; tubular br. in lower lateral region. Breathing and vocal vibration absent at base. Spleen much enlarged (2 years). Expectoration viscid, muco-purulent, somewhat rusty. Appetite good.

Quinine. Pepsine. Cod liver oil. Iodine paint to left chest.

Phthisis.—H. L., æt. 28, Cook. Adm. Oct. 28. Disch. Dec. 23. In H. 56 days. *Relieved.* Dr. Beale, vol. 5, p. 330. Ill 8 months. Pain in chest ; cough. Dulness, and 5 days later br. br. and pectoriloquy under right clavicle ; rhonchus and sibilus over rest of lung ; P. 128, R. 40. 23 days later coarse crep. under right clavicle ; pain under left.

Sesquichloride of iron. Bismuth. Cod liver oil. Blisters under right clavicle.

Phthisis.—C. F., æt. 27, Compositor. Adm. Sept. 11. Disch. Sept. 23. In H. 12 days. *Relieved.* Dr. Beale, vol. 5, p. 286. Prev. ill. 7 months. Cough. Pain in right side. Night sweats. Loss of flesh. Slight hæmoptysis. Flattening, dulness, crepitation, and prolonged expectoration under right clavicle. Crepitation over sup. spinous fossa. Expectoration scanty.

Cod liver oil. Aromatic sulphuric acid, paregoric and bark.

Phthisis.—Emma M., æt. 15. Adm. Sept. 26. Disch. Oct. 17. In H. 21 days. *Much relieved.* Dr. Beale, vol. 6, p. 294. Weakness and emaciation 6 weeks. Cough 14 days. Dulness, cracked pot sound, large crepitation, and increased vocal resonance below left clavicle. Fine crep. over left back ; bronchial br. 9 days later.

Liq. ammon. acetatis. Syrup of iodide of iron and cod liver oil. Iron and calumba.

Phthisis.—Elizabeth T., æt. 21, Works Sewing Machine. Adm. July 14. Disch. Aug. 29. In H. 46 days. *Unrelieved.* Dr. Garrod, vol. 1, p. 175. Hereditary. Subject to cough. Hæmoptysis 16 months ago. Loss of flesh 9 months. Night sweats. Chest resonant ; crepitation over lower front and back of lungs. Appetite bad.

Sulphuric acid and opium. Muriate of ammonia. Bismuth. Tannic acid and conium.

Phthisis.—E. L., æt. 44, Needlewoman. Adm. Sept. 15. Disch. Sept. 26. In H. 11 days. *Relieved.* Dr. Johnson. Ill 2 years. Cough and expectoration. Loss of flesh. Hæmoptysis (½ pt.) 14 days ago and since. Dulness, prolonged expiration, loose crepitation, and bronchophony over left apex. P. 104, R. 40.

Turpentine inhalation. Sulphate of iron and sulphuric acid. Quinine. Cod liver oil and syrup of iodide of iron.

Phthisis.—W. S., æt. 29, Butcher. Adm. Sept. 21. *Died* on Sept. 25. In H. 4 days. Dr. Beale, vol. 6, p. 264. Cough, pain in right chest, and emaciation, 12 months. Worse 8 days. Now, orthopnœa, sputa rusty. P. 124, R. 50. Gurgling and tubular br. under right clavicle ; right lung very dull at back ; br.

br. below mid-scapula; gurgling and tinkling at angle; puerile br. over lower part of right lung in front: elsewhere crepitus.

Carbonate of ammonia and senega. Ether. Brandy. Turpentine stupes.

Phthisis.—W. B., æt. 24, Waiter. Adm. Dec. 20. *Died* on Dec. 29. In H. 9 days. Dr. Garrod. Cough, short breath, and loss of flesh, 15 months. 6 days ago profuse hæmoptysis; occasional attacks during last 8 months. Now very pallid. Dulness and diminished expansion under right clavicle; crepitation over right lung and left sup. spinous fossa. Expectoration purulent, very bloody. Night sweats. P. 100, R. 36. Fresh hæmoptysis, followed by exhaustion and death. P.M.—Cavity size of a small orange in right apex filled with clotted blood; left apex solid, with tubercle; elsewhere scattered tubercles. Emphysema.

Gallic and sulphuric acid. Quinine and sulphate of iron. Turpentine. Turp. inhalation.

Phthisis—Secondary Syphilis.—Rose H., æt. 29. Adm. May 11. Disch. May 18. In H 7 days. Dr. Beale, vol. 5, p. 160. Hæmoptysis 3 years ago; cough since. Loss of flesh. Dulness and prolonged harsh expiration under right clavicle. Vagina ulcerated into rectum. Vesico vaginal fistula. Transferred to surgical ward on May 18.

Hæmoptysis.—M. C., æt. 42, Saleswoman. Adm. July 31. Disch. Aug. 15. In H. 15 days. *Unrelieved.* Dr. Garrod, vol. 1, p. 191. Weak and thin 6 months. Cough 1 month. Night sweats. Hæmoptysis (3 pts.) on morning of adm. Harsh breathing under clavicles. No dulness. P. 68.

Tannic acid, dilute sulphuric acid, and ipecacuanha. Morphia.

Hæmoptysis.—J. H., æt. 59, Hawker. Adm. Dec. 7. Disch. Dec. 30. In H. 23 days. *Recovery.* Dr. Johnson, vol. 18, p. 129. Cough lately, but no other symptoms of phth. Hæmoptysis on 6th and 7th, and again 3 days later. Harsh br. over right post. apex. Some crepitation at bases.

Gallic acid. Turpentine. Iron. Turpentine inhalation.

Asthma.—Eliza K., æt. 34, Married. Adm. June 29. Disch. July 11. In H. 12 days. *Recovery.* Dr. Garrod. Cough and expectoration 1 month. Nightly attacks of dyspnœa and spasmodic cough, relieved by expectoration (14 days).

Sulphuric ether, lobelia, and quinine.

Asthma.—Caroline H., æt. 21. Adm. Oct. 17. Disch. Oct. 24. *Relieved.* Dr. Johnson, vol. 18, p. 59. Hereditary. Always subject to attacks of dyspnœa and cough; now has 2 or 3 daily. Worse in winter. Has emphysema.

Sulphuric ether, lobelia, syrup of iodide of iron, and cod liver oil.

Pertusis—Pneumonia.—Henry T., æt. 8. Adm. Nov. 13. Disch. Dec. 23. In H. 40 days. *Convalescence.* Dr. Garrod, vol. 3, p. 140. Strumous. Cough 2 years, lately whooping. Scanty purulent expectoration. Back of left lung quite dull, breathing absent below, bronchial above, in front vesicular with rhonchus. P. 116, R. 30.

Cod liver oil and syrup of iodide of iron. Belladonna. Conium.

Pertusis—Pneumonia.—Ellen M., æt. 10. Adm. July 14. Disch. Oct. 17. In H. 95 days. *Much relieved.* Dr. Beale, vol. 5, p. 244. Whooping cough on adm. 20 days later spat up ozss of blood. Next day dulness br. br. and crepitation below left scapular angle, with diminished vocal vibration 7 days later. Sputa viscid and tinged with blood. 56 days afterwards br. br. at right scapular angle. On discharge dulness about angle of left and below spine of right scapula. Breathing bronchial over left side, and bronchial with coarse crep. over left back.

Sp. ammon. arom., chloric ether and liq. ammon. acetatis. Syrup of iodide of iron and cod liver oil. Turpentine stupes, and ung. iodinii co. to back.

Acute Laryngitis.—Louisa W., æt. 41, married. Adm. June 8. Disch Aug. 1. In H. 54 days. *Recovery.* Dr. Johnson, vol. 15, p. 231. Syph. sore 2 years ago. Aphonia (6 months) 1½ years ago. Now hoarse 6 months. Dysphagia 1 month. Psoriasis on knees and elbows. On adm. face dusky, much dyspnœa and laryngeal stridor. Glottis reduced by swelling to a small triangular chink posteriorly. Tracheotomy next day. Patient did well; tube removed after 36 days, and wound almost well 5 days later. Slight ulceration of inter-arytenoid fold.

Iodide of potassium. Calomel and colocynth. Leeches over larynx. Mercurialized afterwards. Quinine and iron.

Laryngitis.—Jane E., æt. 32, Hair Net Maker. Adm. Nov. 11. Disch. Dec. 19. *Convalescent.* In H. 38 days. Dr. Johnson, vol. 17, p. 165. O. P. since disch. on Ap. 22, convalescent from laryngitis. Huskiness and, at times, aphonia. Larynx red and congested. Voice natural on 33rd day.

Liq. ammon. acetatis, henbane, chlorate of potass and chloric ether. Quinine and iron. Steam inhalation. Belladonna ointment externally.

Croup.—David M. æt. 3. Adm. Dec. 5. *Died* on Dec. 5. Dr. Beale, vol. 8, p. 8. Delicate. Attacked on morning of adm. Great dyspnœa and exhaustion, cough not brassy, no stridor. Died, exhausted, about 3 hours after adm.

Ipecacuhana emetics. Belladonna and bicarbonate of potass. Belladonna ointment to front, and sinapism to back of neck.

Croup.—Frederick C., æt. 3. Adm. Aug. 24. *Died* on Aug. 25. Dr. Johnson, vol. 17, p. 86. Catarrh and sore throat 19 days. Dyspnœa 4 days. Had treatment. On adm. much dyspnœa, face dusky, P. small and rapid, short cough, crepitation at bases of lungs. Temporary relief from tracheotomy; increase of dyspnœa; exhaustion; death.

Tracheotomy. Brandy and egg. Vin. ipecac.

Aphonia (incomplete).—Eliza K., æt. 20, Servant. Adm. May 20. Disch. July 1. In H. 40 days. *Improved.* Dr. Johnson, vol. 17, p. 43. Weakness and loss of flesh 5 months. Voice husky 6 weeks. Slight cough. Laryngeal mucous membrane anæmic.

Quinine and iron. Ipecac. and squill pill.

Laryngeal Ulceration.—Bridget M., æt. 22, Servant. Adm. June 27. Disch. July 22. In H. 25 days. *Unrelieved.* Dr. Johnson, vol. 16, p. 259. Cough 8 months. Hoarse 5 months. Can only speak in a whisper. Signs of softening tubercle at right apex.

Iodide of potassium and liquor cinchonæ. Chlorate of potass garyle. Hirud. i over larynx.

Laryngeal Ulceration.—Ann M. æt. 30, Dressmaker. Adm. July 23. Disch. Aug. 19. In H. 27 days. *Unrelieved.* Dr. Johnson. Had syphilis. Ill 6 weeks. Aphonia. Glottis congested. Ulceration of left false cord.

Quinine and iron. Cod liver oil. Steam inhalation. Sinapisms to throat.

Laryngeal Ulceration.—Anne E., æt. 32. Adm. June 17. Disch. Aug. 29. In H. 73 days. *Unrelieved.* Dr. Johnson, vol. 16, p. 251. Cough 7 months. Hoarse 3 weeks. Dysphagia and sore throat 10 days. Now complete aphonia, ulcer on right edge of thick red epiglottis, and on right false cord. Swelling of false cords and inter-arytenoidal fold. Tubercle at right apex.

Quinine and sulphuric acid. Cod liver oil. Sol. A. N. to larynx.

Epithelioma of Epiglottis.—William H., æt. 64, Street News Seller. Adm. Sept. 19. Disch. Oct. 14. In H. 25 days. *Unrelieved.* Dr. Johnson. Became

worse after discharge on May 27. On Aug. 10 coughed up an epitheliomatous mass as large as an almond. Voice and deglutition better since.

Chlorate of potass and sesquichloride of iron.

Was re-admitted on Jan. 12, 1864. On Feb. 6 was discharged at his own request. Re-adm. on April 19. The tumour was then much ulcerated on the surface. *Died* suddenly on July 23rd. P.M.—Epiglottis converted into a firm, nodulated epitheliomatous mass, the size of a small apple. The upper part of the left arytenoid cartilage had become atrophied from pressure. The mass was impacted in the glottis. Larynx and tongue healthy. No cancer elsewhere.

Suffocation.—W. D., æt. 15 months. Brought in dead on June 2. Dr. Johnson. No marks of violence. When brought in, trunk warm. P.M.—Slight congestion of cerebral blood-vessels. Verdict on inquest "found dead."

Sore Throat.—S. W., æt. 16, Servant. Adm. May 28. Disch. June 13. In H. 16 days. *Recovery.* Dr. Johnson, vol. 17, p. 17. Prev. ill. 3 days. High fever, yellowish exudation on tonsils. Convalescent on 11th day.

Chlorate of potass, dilute muriatic acid and bark (9 days). Then quinine and sesquichloride of iron. Glycerine and iron to fauces.

Cynanche Tonsillaris.—P. C. æt. 30, Bootmaker. Adm. June 11. Disch. June 17. In H. 6 days. *Recovery.* Dr Garrod, vol. 2, p. 162. First attack. Prev. ill. 7 days. Right tonsil much swelled. Ulcer on left of uvula. Great dysphagia. Convalescent on 11th day.

Liquor ammoniæ acetatis, and mist. alba o. m. (4 days.) Then nitro-muriatic acid and bark.

Cynanche Tonsillaris.—S. S., æt. 30, Hospital Nurse. Adm. Nov. 9. Disch. Dec. 5. In H. 26 days. *Recovery.* Dr. Beale, vol. 6. p. 330. Prev. ill. 3 days. Abscess in left tonsil.

Liq. ammon. acetatis, ether and bark. Dilute muriatic acid and liq. cinchonæ. Wine 4 oz. Iron and glycerine to throat.

Cynanche Tonsillaris.—E. C., æt. 20, Housemaid. Adm. Nov. 18. Disch. Nov. 25. In H. 7 days. *Recovery.* Dr. Garrod, vol. 1, p. 236. Prev. ill. 2 days. Both tonsils swelled. Abscess in left tonsil, and whitish exudation on its surface.

Compound tinct. of lavender and mist. camph. Iron and glycerine to throat.

Ulceration of Throat.—G. C., æt. 24, Shopman. Adm. Aug. 11. Disch. Aug. 27. In H. 16 days. *Recovery.* Dr. Johnson. No syphilis. Prev. ill. 4 days. Chilled while sweating. On adm. tonsils and uvula red and swollen, sloughing of left tonsil and left side of uvula, ulceration of right tonsil. 2 days later slough on post. wall of pharynx; slight delirium.

Carbonate of ammonia, chloric ether and bark. Brandy 9 oz. Afterwards quinine.

Syphilitic Ulceration of Throat.—S. B., æt. 31, Labourer. Adm. June 19. Disch. July 27. In H. 38 days. *Recovery.* Dr. Johnson, vol. 16, p. 249. Sore throat last March. Has lived badly since; discharge from K.C.H. Prev. ill. 6 weeks. Excavated ulcers of tonsils, deepest on left tonsil; back of pharynx ulcerated. Bones of right arm tender and painful.

Chlorate of potass and sesquichloride of iron. Iodide of potassium, liquor cinchonæ, and muriate of morphia. Local application of nitrate of silver.

Syphilitic (?) Ulceration of Throat.—C. P., æt., 25, Wood Carver. Adm. June 6. Disch. July 1. In H. 25 days. *Recovery.* Dr. Johnson, vol. 16, p. 241. Intemperate. Chancre 2 months ago. Sore throat since. Tonsils red, swollen, and ulcerated on surface.

Chlorate of potass and bark (6 days). Then chlorate of potass and sesquichloride of iron.

Ulceration of Throat.—Matilda M., æt. 45, Married. Adm. July 17. Disch. Sept. 2. In H. 47 days. *Recovery.* Dr. Johnson, vol. 19, p. 20. Sore throat 2 months. Aspect cachectic. Uvula and most of soft palate destroyed by ulceration. Left side of throat sloughing.

Iodide of potassium, sesquichloride of iron, and morphia. Belladonna. Local application of nitric acid.

Mercurial Salivation—Stomatitis.—T. S., æt. 27, House Painter. Adm. July 4. Disch. July 18. In H. 14 days. *Nearly well.* Dr. Johnson, vol. 17, p. 177. Never suffered from lead. Subject to gout. Swelling in right groin 3 weeks ago; ten days ago took some pills; 3 days later mouth sore, much salivation. On adm. lower part of face swelled; gums spongy and bleeding; tongue swollen, cannot be protruded far; breath fœtid.

Chlorate of potass, liq. cinchonæ, and chloric ether. Chlorine gargle.

Stricture of Œsophagus.—M. B. æt. 33, Cook. Adm. Oct. 17. Disch. Nov. 16. In H. 30 days. *Relieved.* Dr. Johnson, vol. 17, p. 132. Dysphagia 7 or 8 months, worse last month. Emaciation 1 month. Hoarseness and pain in throat 14 days.

Hot fomentations and steam inhalations. Brandy and beef tea enemata. Afterwards sedatives. Stricture:—dilated by catheters (No. 9–12).

Cancer of Œsophagus—General Paralysis.—H. D., æt. 52, Porter. Adm. July 18. *Died* on Aug. 20. Dr. Johnson, vol. 18, p. 5. Intemperate. Syphilis 35 years ago. 3 months ago loss of power in arms and legs, numbness of hands and feet, headache, occasional vertigo, and temporary unconsciousness. On adm. unable to walk without help; cannot pick up a pin. Radials rigid. P. 56. Marked arcus senilis. 9 days later incontinence of urine. 8 days later dysphagia. 11 days later died suddenly of asphyxia. P.M.—Pericardium universally adherent. Heart fatty. Medullary cancer of œsophagus, forming a tumour the size of an egg, chiefly between œsophagus and trachea; an opening from mass into trachea near bifurcation, through which creamy fluid flowed into trachea. Subarachnoid fluid increased. Brain healthy. Cord not examined.

Iodide of potassium and bark. Strychnia.

Dyspepsia.—W. B., æt. 41, Commissionaire. Adm. Sept. 23. Disch. Oct. 2. *Recovery.* Dr. Johnson, vol. 17, p. 102. Had dysentery in India. Pain and soreness at epigastrium and vomiting 12 days. Coffee-ground matters in vomit. Furred tongue. Bowels confined.

Bismuth. Ammonio-citrate of iron.

Dyspepsia.—R. R., æt. 53, Shopman. Adm. Sept. 29. Disch. Oct. 21. In H. 22 days. *Recovery.* Dr. Johnson, vol. 19, p. 95. Hæmatemesis 8 and 7 years, Jaundice, 2 years ago. Epigastric pain and tenderness, and constipation 2 months. Vomiting 6 weeks, becoming fœcal during last 4 or 5 days. Bowels confined 7 days. Very weak. Epigastrium and left hypochondrium tender. Sarcinæ in vomit.

Enemata of castor oil and warm water. Bismuth and hydrocyanic acid. Oxide of silver and henbane. Sinapisms to epigastrium.

Dyspepsia.—W. H., æt. 45, Labourer. Adm. Oct. 19. Disch. Oct. 24. In H. 5 days. *Relieved.* Dr. Garrod, vol. 3, p. 90. Occasional vomiting after meals, 4 years. Sudden pain at epigastrium 2 years ago while lifting a heavy

weight. Ten weeks afterwards hæmatemesis, melæna, and return of pain. Epigastric pain 1 week. Vomiting 5 days. Tongue white.

Bismuth and calumba.

Dyspepsia.—Selina P., æt. 48, Servant. Adm. June 10. Disch. July 8. In H. 28 days. *Nearly well.* Dr. Beale, vol. 5, p. 196. Pain and soreness of epigastrium, 2 months. Waterbrash and loss of appetite, flesh, and strength, 1 month. Vomiting; anæmia.

Bismuth and dilute hydrocyanic acid. Henbane, sesquichloride of iron, and dilute hydrocyanic acid.

Dyspepsia.—F. M., æt. 61. Monthly Nurse. Adm. Sept. 26. Disch. Oct. 28. In H. 32 days. *Relieved.* Dr. Beale, vol. 6, p. 280. Pain across epigastrium, vomiting, and hæmatemesis 2½ months ago. Jaundice 14 days later. Loss of appetite, sour risings, waterbrash, constipation. Moveable painless tumour in left hypochondrium, probably connected with left lobe of liver.

Mist. Rhei. Co.

Dyspepsia—Partial Peritonitis?—H. G., æt. 44, Probationer, K.C.H. Adm. July 12. Disch. Sept. 25. In H. 75 days. *Recovery.* Dr. Johnson, vol. 16, p. 269. Pain after food and waterbrash 6 weeks. On day before adm. great pain in abdomen and vomiting. Some blood in vomit. Epigastrium extremely tender. Tongue furred. Appetite bad. P. 96. Free from acute symptoms 8 days later. Relapse 16 days later.

Bismuth hydrocyanic acid and muriate of morphia. Citrate of iron, hydrocyanic acid, and calumba. Iron and compound galbanum pill.

Hæmatemesis.—Ellen C., æt. 28, Married. Adm. Nov. 23. Disch. Dec. 16. In H. 23 days. *Recovery.* Dr. Garrod, vol. 19, p. 180. Not subject to vomiting or abdominal pain. On day of admission, soon after lifting a tub of water, pain in left side, faintness and copious hæmatemesis. Hæmatemesis recurred once on night of 24th, twice on 25th, and once on night of 25th. Much blood passed by bowels during this time.

Gallic acid. Ice. Enemata of brandy and beef tea. Turpentine. Sesquichloride of iron and quinine.

Hæmatemesis.—L. A., æt. 47, Cook. Adm. Sept. 18. Disch. Sept. 26. In H. 18 days. *Recovery.* Dr. Johnson, vol. 19, p. 97. Intemperate. Four days ago, faintness followed by vomiting of blood. No dyspeptic symptoms or fresh hæmatemesis.

Turpentine and chloric ether. Afterwards iron and columba.

Pyloric Obstruction.—A. B. S. R., æt. 69. Adm. Sept. 23. Disch. Oct. 5. In H. 12 days. *Very much relieved.* Dr. Beale, vol. 5, p. 290. Father died of cancer. During last month pain and soreness of epigastrium, much flatulence, vomiting, and loss of flesh. Appetite bad. Stomach much dilated. Vomit contains sarcinæ. Urine 1-12th albumen. No tumour.

Bismuth and paregoric. Creosote.

Pyloric Obstruction.—J. C., æt. 49, Spurmaker. Adm. Nov. 3. Disch. Nov. 18. In H. 15 days. *Much relieved.* Dr. Johnson, vol. 17, p. 141. Formerly drank large quantities of beer. Vomiting, loss of appetite, flesh, and strength, 18 months. Has vomited twice or thrice daily for last six weeks. Very pale and thin. Stomach enormously dilated. Vomit contains sarcinæ. No tumour.

Strychnia and dilute muriatic acid. Strychnia and sesquichloride of iron. Purgatives.

Cancer of Stomach.—D. H., æt. 47, Cellarman. Adm. June 11, again Sept. 24 and Nov. 7. *Died* on Nov. 11. Dr. Johnson, vol. 17, p. 87. Loss of flesh

and strength, and pain at epigastrium after food, 2 months. Loss of appetite, 6 weeks. On adm. a hard moveable tumour the size of a large orange in umbilical region of abdomen. Occasional vomiting. In H. 37 days. On re-adm., Sept. 24, more emaciated; tumour larger and more tender. In H. 50 days. Re-admitted on Nov. 7. Abdomen tympanitic; vomiting daily. Four days later severe abdominal pain, followed by collapse and *death*. P.M. Perforation of ascending colon. Recent peritonitis; cancer of stomach, involving colon and part of small intestine, but not stricturing pylorus.

Quinine. Iron and hydrocyanic acid. Bismuth. Opium.

JAUNDICE.—A. P., æt. 32, Blacksmith. Adm. Nov. 9. Disch. Dec. 16. In H. 37 days. *Recovery.* Dr. Beale, vol. 5, p. 349. Subacute rheumatism 4 months ago. Urticaria 14 days ago. Jaundice 10 days. Lemon coloured, languid, sleeps badly. Motions clay coloured. No hepatic pain, tenderness, or enlargement. Right hip and shoulder tender.

Iodide of potassium, bicarbonate of potass, and decoction of bark (12 *days*). *Then dilute muriatic acid and decoction of bark. Hydrochloric acid lotion. Grey powder and rhubarb.*

JAUNDICE.—Frances W., æt. 34, Married. Adm. July 17. Disch. Aug. 1. In H. 15 days. *Recovery.* Dr Garrod, vol. 2, p. 232. Five months pregnant. Ill 8 days. Nausea, loss of appetite, jaundice. Motions white. No hepatic pain, tumour, or tenderness.

Castor oil. Quinine and sulphuric acid.

JAUNDICE.—E. B., æt. 20, Servant. Adm. Sept. 7. Disch. Sept. 23. In H. 16 days. *Recovery.* Dr. Garrod, vol. 2, p. 271. Jaundice, vomiting, and constipation, 1 week. Skin deep yellow; pain and tenderness across upper part of abdomen. Liver not enlarged. Motions white. Soft systolic bruit at base of heart.

Sulphate of magnesia, bicarbonate of potass, and columba. Castor oil.

JAUNDICE.—W. T., æt. 71, Shoemaker. Adm. Sept. 8. Disch. Sept. 23. In H. 15 days. *Recovery.* Dr. Johnson, vol. 19, p. 56. Loss of flesh and appetite 9 months. Lately worried at being out of work. Epigastric pain and soreness 10 days. Jaundice 4 days. Motions pale; no enlargement of liver.

Dilute nitro-muriatic acid and decoction of bark.

JAUNDICE.—E. H., æt. 26, Milliner. Adm. Oct. 14. Disch. Dec. 2. In H. 49 days. *Recovery.* Dr. Garrod, vol. 3, p. 81. In K. C. H. last April with albuminuria. Skin light yellow; slight hepatic tenderness; motions pale; urine 1-8th albumen; tongue coated; venous murmur in neck; albumen persistent.

Sal-volatile, chloric ether and taraxacum. Quinine and bicarbonate of potass. Iodide of potassium and bark.

JAUNDICE.—Emma S., æt. 10. Adm. Sept. 22. Disch. Sept. 30. In H. 8 days. *Recovery.* Dr. Johnson, vol. 17, p. 105. Jaundice and dry cough 4 days. Bowels confined. Motions pale. Pain in lower part of chest. No hepatic tenderness.

Sulphate of magnesia, sp. ammon. arom., and decoction of bark (6 *days*). *Iron and quassia. Aperients.*

JAUNDICE.—T. G., æt. 39, Labourer. Adm June 8. Disch July 11. In H. 33 days. *Recovery.* Dr. Beale, vol. 5, p. 192. Had gout twice; cough 3 years. Prev. ill. 3 days. Out of work 2 days before. Skin lemon coloured; motions pale; no enlargement of liver.

Dilute muriatic acid and quinine. Bicarbonate of potass and chloric ether. Aperients. Muriatic acid lotion.

JAUNDICE.—Mary B., æt. 56. Adm. Oct. 30. Disch. Nov. 21. In H. 22 days. *Recovery.* Dr. Beale, vol. 6, p. 328. Subject to sick headache. Prev. ill. 1 week.

Retching. Loss of appetite. Skin deep lemon colour. Considerable itching. No pain, tenderness, or enlargement of liver. Tongue furred.

Dilute muriatic acid. Muriatic lotion applied over liver.

JAUNDICE.—M. A. F., æt. 18, Straw Bonnet Maker. Adm. June 24. Disch. July 4. In H. 10 days. *Recovery.* Dr. Beale, vol. 5, p. 208. Subject to headache. Was frightened 24 days ago and had a fit. Became jaundiced next day. Loss of appetite, constipation, much itching of skin, aching pain over liver for last week. Skin pale lemon colour. Faint basic systolic bruit.

Hydrochloric acid lotion. Pil. col. co. and mist. alba.

JAUNDICE.—W. C., æt. 19, Blacksmith. Adm. Oct. 3. Disch. Oct. 21. In H. 18 days. *Recovery.* Dr. Johnson, vol. 18, p. 19. Drinks spirits freely. Jaundice 12 days. Languor and weakness, nausea and loss of appetite. Skin dark lemon colour; much itching; motions pale; slight pain and tenderness in right hypochondrium; no enlargement of liver.

Podophyllin and colocynth. Dilute nitro-muriatic acid, chloric ether, and bark. Brandy 2 *oz.*

JAUNDICE.—S. J., æt 21, Waitress. Adm. Sept. 3. Disch. Sept. 11. In H. 8 days. *Recovery.* Dr. Johnson, vol. 19, p. 41. Far advanced in pregnancy. Lately deserted by husband. Jaundice three weeks. Motions at first clay coloured; vomiting; skin deep yellow; much itching; sleeplessness; no hepatic enlargement or tenderness; tongue red. Transferred to lying-in ward on Sept. 11; nearly free from jaundice.

Sulphate of magnesia, dilute sulphuric acid, chloric ether, and sp. meath. pip. Morphia.

JAUNDICE.— A. W., æt. 34, Married. Adm. Oct. 14. Disch. Nov. 4. In H. 21 days. *Recovery.* Dr. Beale, vol. 6, p. 310. Pregnant 4 months. Bowels much confined since. Was "upset" 14 days ago. Soon afterwards headache, loss of appetite, pain round upper part of abdomen, and jaundice. Languid and wakeful. Skin lemon coloured; itching; appetite bad; motions pale: oxalate of lime in urine.

Dilute muriatic acid. Castor oil. Muriatic acid lotion applied over liver.

*JAUNDICE.—W. F., æt. 34, Coachman. Adm. April 27. *Died* on May 5. In H. 8 days. Dr. Johnson, vol. 16, p. 195. Temperate. Vomiting and headache 1 month. Ill 14 days with hepatic pain, languor and jaundice. On 10th day P. 140, and violent delirium, subsiding after stimulants. On adm. slightly jaundiced, edge of liver 2 in. above umbilicus, delirium, subsultus, bowels confined. On 16th day sleepless, petechiæ on trunk and arms, motions pale brown passed involuntarily, tongue dry, brown and fissured. On 21st day dulness, bronchial breathing, and bronchophony over left base, crepitus over right base, increased subsultus, other symptoms as before. *Death* on 22nd day. P.M. Common gall duct the size of a forefinger, its lower end blocked by a gall stone as big as a marble. Liver weighed 4 lbs. 3 oz., ducts distended with pale bile. Interior of gall bladder ulcerated, containing one gall stone. Lower lobes of lungs hepatized, also back part of left upper lobe.

Quinine. Purgatives. Chlorodyne.

*JAUNDICE.—E. H., æt. 33, Married. Adm. May 14. *Died* on June 9. In H. 26 days. Dr. Garrod, vol. 1, p. 104. Jaundice 15 years ago. Became jaundiced 6 days ago, after 3 days pain in right hypochondrium. On adm. deeply jaundiced; liver tender, not enlarged; motions faintly bilious; urine deep yellow; vomiting. Coma on 21st day of jaundice. Death on 24th day. P.M. Liver 2 lb. ¾ oz.; dark red, with large atrophied patches of a gamboge tint. Cystic and bile ducts pervious. Thirteen tetrahedral stones in gall bladder. Two inches of fat on abdomen.

Treatment much varied. Sulphate of magnesia, belladonna, calomel, asafœtida, podophyllin taraxacum.

JAUNDICE– GOUT.—F. M, æt. 44, Publican. Adm. March 9. Disch. April 4.

if it were within the sarcolemma (*c*), but the fibres proceeding from each extremity render such a position impossible. The relation of these nerve-nuclei to the sarcolemma is seen at *l* in profile. The nuclei, as well as the fibres for a certain distance, often adhere to the sarcolemma very firmly; but in the thin mylo-hyoid muscle the course of the fibres over or under, but always *external*, to the muscular fibres, may be readily traced if the muscular fibres be separated slightly from each other, as represented in the drawing.

At *d* fine nerve-fibres accompanying the fine fibre continued from the dark-bordered fibre, as described in the "Philosophical Transactions" for 1862, are represented. Such fibres are also seen at *e* and *f*.

m, *n*, and *o*. Dark-bordered fibres, with nuclei near their distribution. *m* Would probably pass over sixty or seventy muscular fibres, and *n* over perhaps twenty, before it divided into fibres as fine as those seen at *b*, *e*, *f*, *l*.

p. A very fine capillary vessel with a nerve-fibre running close to it.

q. A bundle composed of six very fine nerve-fibres near their distribution. These fibres exhibit a very distinctly beaded appearance, which is also observed in many other fine fibres in different parts of the specimen.

Traces of connective tissue are seen in all parts near the fine nerve-fibres and around the muscular fibres. Here and there some very fine connective tissue-fibres, which were not altered by acetic acid, are represented. These represent the remains of fine nerve-fibres, which existed in a state of functional activity at an earlier period.

The drawing, with the exception of the position of the nerve-fibre (*o*) above mentioned, is an actual copy from nature. The relative position of the muscular fibres, the form and general character of the so-called nuclei, and the position and size of the nerve-fibres and their nuclei, have been carefully preserved.

The very fine nerve-fibres have been traced, in so many instances, from one trunk to another ramifying at a very considerable distance, that it is almost certain that no true terminations or ends exist.

Plate XIII.

To illustrate Dr. Beale's observations upon the controversy relating to the Distribution of Nerves to Striped Muscle, page 160.

Fig. 1.—Dark-bordered fibres. The nucleated dark-bordered fibre *a* is seen to divide into two branches. The one

passing in the division which diverges to the right, rapidly becomes so very fine that it can hardly be followed as it runs parallel with the other dark-bordered fibre and in the same sheath with it (under *d*). This very fine fibre, in direct continuity with the dark-bordered fibre, cannot be distinguished from the fine fibres usually regarded as connective tissue seen in the sheath of the nerve on the left side of the specimen (at *b*), which leave the trunk and assist in forming a separate branch at *c*. From the pectoral. × 700. The finest nerve-fibres are really much finer than is generally supposed.

Fig. 2.—Dark-bordered nerve-fibre with fine fibres on one side of it. These fine fibres appear in one part like the outline of the tubular membrane, and are usually considered to be connective tissue, but they are seen to give off branches which, with other fine fibres of the same character, form networks.

Fig. 3.—Fine fibres from the sheath of the nerve, dividing and subdividing on the surface of two elementary muscular fibres. From muscle of the neck of the frog. × 700 diameters. Compare the appearance of the fibres represented at *b*, *b* in the drawing, with fibres *b* in figs. 1 and 5, which undoubtedly come from true dark-bordered nerve-fibres.

Fig. 4.—Very fine nerve-fibres on the surface of the muscular fibre. From the pectoral of a young frog. Several of these fibres can be followed to the edge of the muscular fibre, where they are lost. × 1800.

Fig. 5.—Young muscular fibre from the pectoral muscle of the frog, near one of the oval swellings. At *a* and *b* are seen the fibres which are continuous with the dark-bordered fibres. These are gradually lost amongst very fine fibres. The compound branches divide and subdivide on the surface of the muscular fibre. × 700.

Fig. 6.—Fine dark-bordered fibre, and fine nerve-fibres which are generally looked upon as fibres of connective tissue in the sheath of the nerve, with nuclei. From muscle of the neck of the frog. × 700.

Plate XIV.

To illustrate Dr. Beale's observations upon the controversy relating to the Distribution of Nerves to Voluntary Muscle, page 160.

Fig. 7.—Division of dark-bordered fibre with fine fibres ramifying in the sheath of the nerve. From the pectoral muscle of the frog. × 700. The fine fibres, it will be

observed, break up into bundles composed of still finer fibres. These can be followed for some distance, and at last, from their extreme tenuity, they are lost.

Fig. 8.—Division of dark-bordered fibre on surface of muscular fibre. Pectoral muscle of the frog. The fibres resulting from the subdivision of the dark-bordered fibre may be traced for some distance amongst the very fine fibres.

Fig. 9.—Division of dark-bordered fibres and formation of networks of pale compound and very fine fibres. Also from the pectoral of the frog.

Fig. 10.—Branching and division of dark-bordered nerve-fibre near terminal branches, and bundles of fine fibres. Several nuclei are seen in connection with both sets of fibres. From the bladder of the frog. × 700. At *a* the dark-bordered fibre becomes resolved into several fine fibres. It is in this way that the dark-bordered nerve-fibre invariably 'ceases.' It is not the "axis cylinder" which is alone continued, as is usually stated, but the substance of the fibre breaks up to form a bundle of very delicate fibres. This bundle divides and subdivides, and, with bundles from other fibres, helps to form a network.

Plate XV.

To illustrate Dr. Beale's observations upon the controversy relating to the Distribution of Nerves to Striped Muscle, page 160.

Fig. 1.—Eight muscular fibres seen in section, showing the manner in which, according to Kühne, the nerve-fibre perforates the sarcolemma, and comes into close relation with the contractile tissue. M., Muscular fibres; N., nerve-fibres; *b*, collection of nuclei of the nerven-hügel.

Fig. 2.—Part of another drawing more highly magnified, to illustrate the same point. Also after Kühne.

Fig. 3.—Copy of a schematic figure reduced one-half. After Kühne. This figure illustrates the latest inferences of this author upon the termination of motor nerves in the muscular fibres of amphibia and mammalia, including man. A, axis cylinder and its expansion as an end-plate; *a a*, white substance; *b*, sheath; *c*, nuclei of the sheath; *d*, a nucleus of the same kind in the membrane of the nerven-hügel; B, contents of the nerve eminence or nerven-hügel, a granular substance (Protoptasm?); C, the clear nuclei of the nerve eminence, with their large nucleoli; D, solitary large

nucleoli (?) in the granular contents of the nerve eminence; E, nucleus of the muscle; F, contractile tissue with the sarcous elements; G, sarcolemma.

Fig. 4.—Muscular fibre from the maggot. (L. Beale.) The sarcolemma is removed from the upper part. A bundle of nerve-fibres is seen crossing the muscle, from which finer but still compound fibres proceed. To the right a branch may be traced downwards to the sarcolemma, which is drawn out so as to form a little eminence (nerven-hügel) at the point where the nerve comes into contact with it. The nerve-fibre divides at this point into finer fibres, which ramify upon the surface of the sarcolemma, and are adherent to it. Above and below the nerve-fibre are numerous branches of tracheæ, which also ramify upon the sarcolemma, and are intimately adherent to it.

Fig. 5.—Represents one of these so-called nerve eminences more highly magnified. Immediately within the fold of the sarcolemma is one of the large nuclei of the muscle. The nerves forming the fine nerve-trunk (*b*) pass, some on the anterior, and some on the posterior, surface of the sarcolemma, as represented in the drawing. *a, a,* are branches of tracheæ.

Plate XVI.

To illustrate Dr. Beale's observations relating to the Distribution of Nerves upon Striped Muscle, page 160.

Fig. 1.—A nerve-tuft or swelling. After Kölliker. This is described as "a nodule or swelling of a muscular fibre (or rather fasciculus), with a large nerve-fibre, nerve-tuft, and nuclei; highly magnified."

Fig. 2.—A portion of one of the nerve-tufts or swellings from the breast muscle of the frog. Prepared according to the process alluded to in page 164. The dark-bordered fibres are seen to divide and subdivide into very fine fibres, which run amongst the developing muscular fibres which are distinctly seen in the drawing. The transverse markings can be discerned in many places.

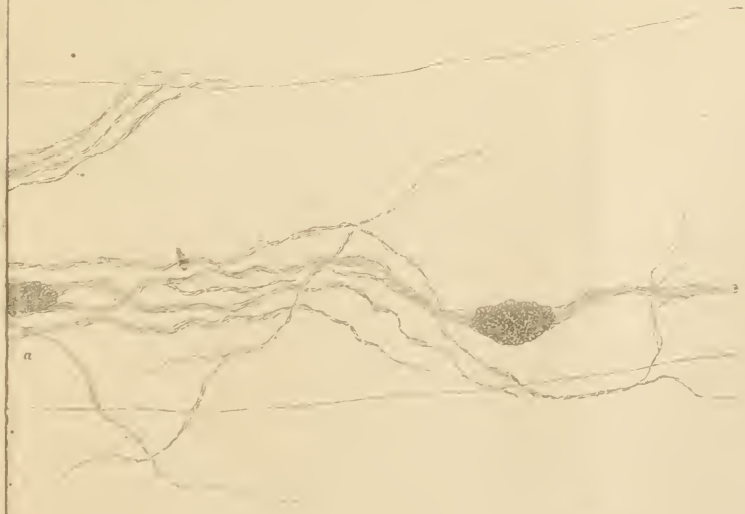
At *a* is a portion of a young muscular fibre, to show the manner in which the contractile tissue is formed by the agency of the "nuclei," or masses of germinal matter.

Fig. 3.—From the same, more highly magnified. The muscular fibres and their nuclei are omitted, in order to show the subdivision and mode of ramification of the nerve-fibres more distinctly. The fine nerve-fibres ramifying in the sheath of the nerve are represented in figs. 2 and 3.

× 70.

Fig. 7.

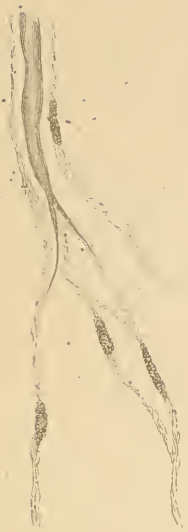
× 70.



× 180.

PLATE XIV.

Fig. 7.



× 700.

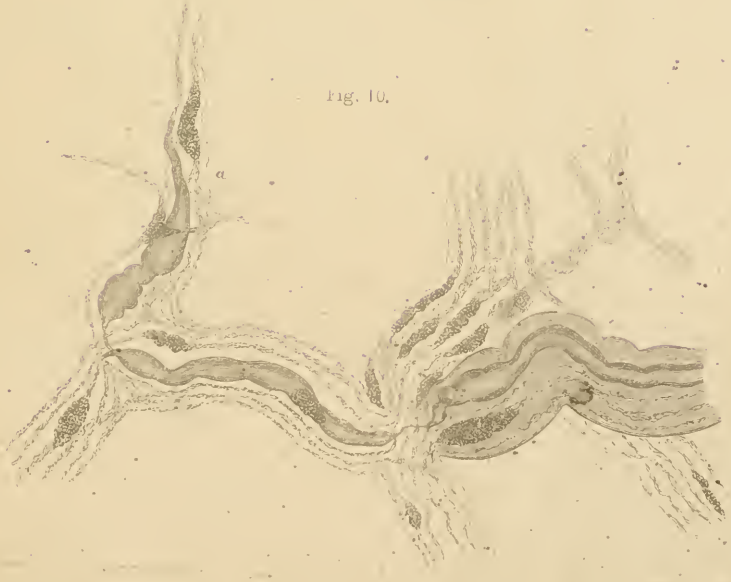
Fig. 8.

× 700.

Fig. 9.

× 700.

Fig. 10.



× 700.

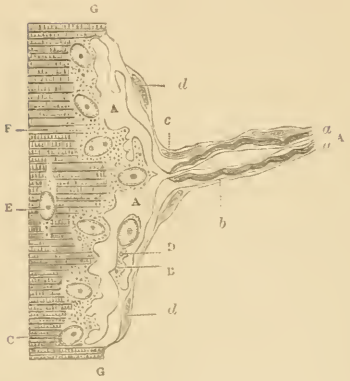
Fig. 1.

Kühne.

Fig. 2.

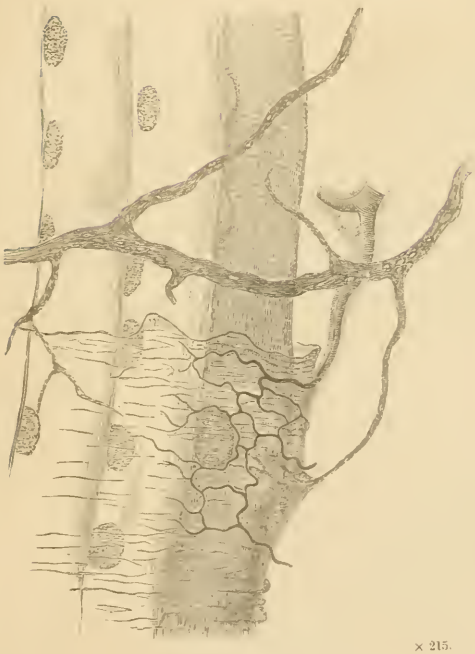
Kühne.

Fig. 3.



After Kühne.

Fig. 4.



× 215.

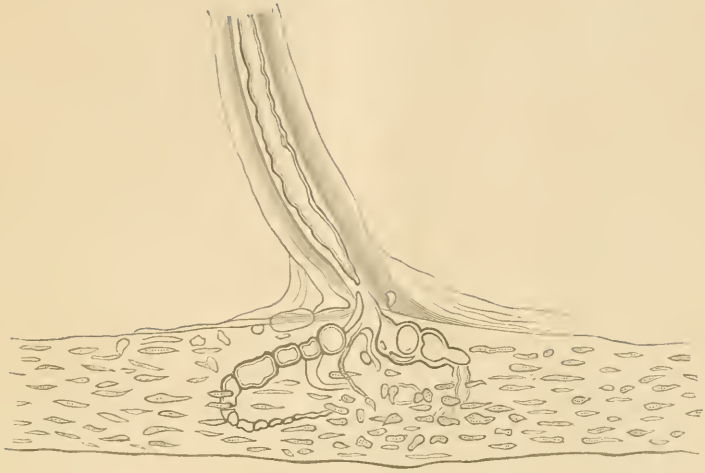
From the insect. L. S. B.

Fig. 5.

× 700.

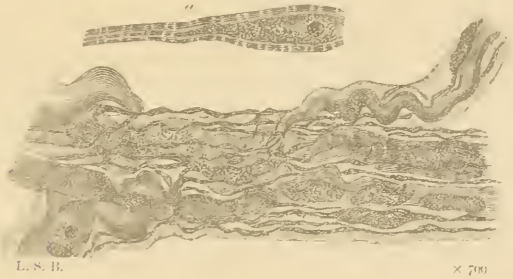
From the insect. L. S. B.

Fig. 1.



Kölliker.

Fig. 2.



L. S. B. × 700

Fig. 3.

L. S. B. 1000.

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Part XVI., with Index to the whole work, concluding Vol IV., of the Archives will be published shortly: The following papers have been received, and will appear in the next part :—

On a Case of Paraplegia, by J. Lockhart Clarke, F.R.S.—On Muscular Power, by H. F. Baxter.—On the Development of Morbid Bone, by Dr. Roudneff.—On the Moderating Centres of the Reflex Function of the Spinal Cord, by Alexander Herzen, M.D.—On a Curious Structure in connection with an Ovarian Cyst, by Dr. Ritchie.—Fungi Developed in the Pyramids of the Kidney, by Dr. Tonge.—A Review of Dr. Dean's work on the Medulla Oblongata, by W. B. Kesteven, F.R.C.S.—Microscopic Examination of a small moveable Subcutaneous Tumour, by W. B. Kesteven, F.R.C.S.—Cases treated in the Medical Wards of King's College Hospital, by Dr. Tonge, concluded. With numerous plates.

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